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Table of Contents

CANCER AND TUBERCULOSIS: X. AN APPROACH TO THE PROBLEM OF THE STOMACH.[1]

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CANCER has created an inferiority complex in the minds of most medical men over the age of fifty. They have experienced a very large measure of ill success in dealing with the disease; every theory of its nature has in turn been discredited by additional knowledge; new remedies excite no interest, because not one has made good even a part of its claims; worst of all, the immense amount of research concentrated on the problem for more than thirty years has thrown little direct light on the causation of the disease in man. If, however, the direct attack has yielded poor results, there are indirect methods leading to subsidiary facts which may be significant. I propose to examine certain features peculiar to a number of sites, and these peculiarities seem to lead to new conceptions of cancer, especially in its most common site—the stomach.

The line of investigation on which I have been working for the past ten years was suggested by the reciprocal variations in the death rates, the decline in that from tuberculosis being accompanied by an almost equal increase in that from cancer. In 1925 I made a provisional explanation of this fact by showing that not only did the numbers balance each other, but that by following out the life history of any given group of young adults, certain percentage rates were balanced in such a way as to point to a close relationship in the conditions under which the two diseases arose. The completion of the ten-year period 1921–1930 enabled me to show that phthisis and cancer have uniformly caused the deaths of 22·5% of the young adults that reached the age

[1] Publication of the Fletcher Cancer Research Fund.

group 15–25 in each successive census of 1841, 1851, 1861 and 1871, and that up to the age of 65 the same group for 1881 also falls in line. It thus appears certain that in England for the past hundred years the deaths from cancer have been restricted to the moiety of the 22·5% which has not died from tuberculosis. In other words, under existing conditions of life and of exposure to the exciting cause, there is a definite limit to the number of people who are susceptible to cancer.

This proposition might be admitted if it ran "who are susceptible to tuberculosis", but the data just referred to show that as phthisis and cancer combined have been limited to the 22·5% of the population above the age of 15 years and the former of these diseases takes its toll at the earlier ages, so cancer, its successor, is at present restricted to the portion of the 22·5% which has escaped phthisis.

The Problem of Site Incidence.

In 1922 I compiled from Hoffman's "Mortality from Cancer throughout the World" a list of countries, chiefly European, showing that when taken together cancer of the digestive system and skin in pre-war periods usually accounted for about 90% of the total incidence of the disease in men; and that the digestive system, breast and uterus provided a similar percentage in women. The distribution of the disease among the different sites varied greatly, of which the following examples are selected as showing the proportions of the total incidence which fell upon the digestive system: for men—Bavaria, 84%; England, 75%; Italy, 60%; for women—Holland, 67%; England, 47%; and United States of America, 40%.[1]

Post-war observations have emphasized the wide distribution of such variations in the site incidence, and the matter was considered at Geneva to be one of international importance. Experts of the League of Nations Health Organization spent the five years 1922–1927 on an investigation of certain national statistics. Their principal work dealt with the causes of the very notable difference in the site incidence of cancer of the breast and uterus in different countries.

> Every conceivable method of analysis has failed to produce any single clear-cut explanation of the divergencies in the national mortalities from these forms of cancer in the countries studied.[2]

The Registrar-General in 1927[3] pointed out that in males cancer mortality shows very definitely the same type of social distribution as that from phthisis. Taking the average of all males as 100, that of Social Class I was 80, and the maximum was reached in Social Class V at 123, or a ratio of approximately 3 : 2. Moreover, cancer of the skin, lip, tongue, œsophagus and stomach were in the ratio of 50 for Class I and 140 for Class V. Assuming for the moment that cancer is always caused by various forms of definite chronic irritants, these observations may be carried a step further by evidence from the occupations. Certain sites, presumably those most subject to the special form of irritation, bear the brunt of the attack. Thus among sweeps and cotton spinners in the census period 1921–1923 cancer of the skin formed 24% of the total deaths from malignant disease; whereas in the deaths in the general male population aged 16 and upwards the skin accounted for only 3·2% of these deaths. But in regard to the lip, tongue, œsophagus and stomach we find that in sweeps, the clergy, clerks, cotton spinners and bankers these sites show the lowest incidence, contributing about 25% of the total deaths from cancer; whereas in farm, dock and unskilled labourers, coal miners and costermongers, the proportion contributed by these sites rises to 40%. It appears, therefore, that the excessive incidence of skin cancer has accounted for so many sweeps and cotton spinners that the average conditions of irritation could not secure as large a number of susceptible men for the sites in the upper alimentary canal as their social position would lead one to expect. In other words, most of the men who in 1921 were susceptible to cancer in these two occupations appear to have died of that disease.

A similar trend of evidence comes from several other sources. Thus in regard to the breast we find that in England and America cancer is controlled to the extent of two-thirds of the cases by the disuse of the organ.[4] Women who have never nursed a child contribute two cases out of every three; and the more children, the less cancer.[5] Both these countries have a low birth rate. Examples with much lower breast rates than the English-speaking peoples are Switzerland, Bavaria, Holland, Norway, Italy and Japan. These have a much higher rate for both the stomach and intestines, which reaches its maximum in Norway and Holland. Japan, Italy and Bavaria have high birth rates, and in Japan the ancient custom of nursing a child for two years is still maintained. The periodic functioning of an organ whose cells are waiting to respond to the normal call to activity may be the decisive factor which explains the influence of disuse. But especially in the case of Norway and Switzerland we find a low birth rate, a low incidence of cancer of the breast, and an excessive incidence of cancer of the stomach. It seems, therefore, as if the stomach in these countries holds a similar position among the sites as the skin does in sweeps and cotton spinners in England. Hence it appears that in Norway and Holland the unknown irritant has the choice of either the breast or the stomach, and for some reason selects the latter organ. In the case of the breast, it is generally believed that the usual preliminary to cancer is the chronic irritation caused by retained secretion of the epithelial cells, whether this irritation goes through an inflammatory stage or not. Such secretion may give rise to unsaturated fatty acids which appear to be suitable intermediate steps to the production of growth stimulating substances, perhaps carcinogenic in their action. On paper, therefore, it appears easier for an irritant to act on the mamma than on the stomach.

Internal Irritants.

The evidence that a focus of chronic irritation is in some unknown way the cause of cancer has accumulated from both clinical and experimental sources during the past twenty-five years. It has

been shown that such diverse factors as tar, mineral oils, soot, heat, cold, radium and X rays are capable of setting up cancer of the skin, and that the most obvious factor which unifies these very different agents is the manner in which they all bring about a lymphocytosis around the spot where the change to malignancy will occur. Clinically, the rôle of chronic irritation has been rendered more and more definite by detailed information on certain internal forms of the disease which are not seen in England or Australia, but are common in other countries. Thus, betel cancer affecting the cheek, gum or tongue associated with the national habit of betel chewing is common only in Ceylon and a strip of the adjacent south-west coast of India. In these regions it constitutes from 45% to 75% of all cases of cancer treated in the hospitals. It is practically restricted to certain districts, and affects only the poorest and most ill-nourished of the people. These correspond to Class V of the Registrar-General, except that they are much nearer the starvation line. The exciting cause seems to be the more irritating qualities of varieties of both lime and tobacco which form part of the quid, and which are used only among the poorer classes. Betel cancer is almost unknown in other parts of India, although the habit is very widespread.[6]

Although the association with a single irritant is not so clear in European countries, yet it is generally held that "buccal and lingual cancer is almost always referable to one or all of three factors—syphilis, tobacco and decayed teeth".[7] The presence of any one of these factors as a chronic irritant produces conditions involving well marked lymphocytic infiltration of the submucous tissues—a condition which I regularly find in mice inoculated with tubercle bacilli in the neighbourhood of lesions of the skin, alimentary canal, or mammary gland. The clinical and experimental evidence therefore shows that these precancerous conditions are closely related.

Recognizable points of irritation are by no means confined to the skin and mouth. A good example in the internal organs is bilharzial cancer, which may be called the national type in Egypt. In the case of males it forms fully 13% of all cases of cancer. The irritation is produced by the eggs of a trematode worm which lodges in the radicles of the veins of the portal system, whence the eggs are found in the submucous tissue of the bladder and rectum, and malignant papillomata result.

The origin of primary tumours of the liver as the result of parasitic infection has been established by Bullock and Curtis. They find that infestation of rats with the tapeworm of the cat produces tumours, both innocent and malignant, in about 10% of the rats. Most of these tumours are found in the liver, but about one-fifth of them select other sites, including the skin, mammary and lymphatic glands and bones. As some of them arise independently of any aberrant cyst, it is possible that the irritant may be the bacteria which the larvæ carry with them from the interior of the rat's intestine when they penetrate its walls. This point is being investigated at the present time.[8]

Analogous facts suggest the explanation of primary cancer of the liver in countries where a large proportion of the people are known to harbour intestinal parasites. Instances may be cited from Asiatic Russia, southern India, Sumatra, and many parts of Africa. In Sumatra, native and Chinese coolies work under similar conditions on the plantations, but the proportional distribution of the site incidence is very different. In the Chinese the liver contributed 28% and the stomach 19%. In the natives the liver 55% and the stomach 1%.[9] In the liver, primary cancer is usually associated with cirrhosis, a condition which suggests the presence of a definite irritant of bacterial, parasitic or other origin.

The uterus with its adnexa is the second site in order of frequency in the women of many countries, coming next in this respect to the stomach. Three-fourths of the cases are at or external to the cervix, and the remainder in the ovary, tube, or body of the uterus. In the former group, 90% of the women have borne children, and there is almost invariably some sign of injury to the cervix. Hence the sequence of injury, invasion by polymorphonuclear organisms, proliferation during repair, occlusion of mucous ducts with retention of secretion and isolation of epithelium, can often be inferred, and in this way these changes can be linked to the changes in the skin in experimental tar cancer. But in the second group there is usually no question of injury, and although physiological proliferation is part of the function of the ovary, this organ is one of the less common sites of cancer. As to the exciting cause of cancer of the ovary, we are completely in the dark. Developmental anomalies are regarded as important precancerous conditions, and it has been urged that the not infrequent bilateral symmetry is evidence against the presence of an external agent. But bilateral distribution is no evidence against bacteria, rather the reverse, for tuberculosis of certain abdominal organs in both sexes is often bilateral.

Irritants and Cancer.

The variation in the predominance of one site in different countries, sexes and occupations is evidence that cancer is not essentially bound to any single type of epithelium. In regard to other tissues there is similar evidence. Thus primary malignant disease of the bones appears to be much more frequent in males than females, but secondary deposits are very much more frequent in women, because the ribs and other bones are easily invaded from a primary tumour of the breast. It is true that the agent is different—a transplanted cell in the secondary and the unknown irritant in the primary tumours, but there does not appear to be a special proclivity in either sex. The analogy appears to be with the more frequent occurrence of pyogenic affections of the skin and bones in males, which may perhaps be explained in terms of greater liability to infection.

The widely different forms of carcinogenic irritation taken in conjunction with this diversity of sites suggest that the apparent selective action of cancer is a matter of ease of access of the specific irritant in an individual whose tissues are prepared to make the necessary response.

From the foregoing survey it appears that all the known causes of cancer may be classified as irritants, but some of them which produce a national type of

cancer in one country may be of small importance in other parts of the world. This difference is not due to any inborn racial peculiarities of the two peoples, but to a difference in their habits. The number of the important sites at which cancer appears in response to known irritants is steadily increasing, and the identification of these irritants is rapidly reaching the point at which it may be claimed that chronic irritation of some kind is the definite forerunner of cancer. In some of the instances which have been briefly discussed in this paper the irritant is obvious, and in others, such as the mamma and uterus, the chronic irritation probably involves the presence of microorganisms as the explanation of the initiation of the changes that lead to malignant disease. The distinction must be stressed between what happens within the cell and the changes that take place outside it, that is, between the direct and the indirect causes of cancer. Carcinogenic chemical substances or a virus may be the direct cause of proliferation of the cells, but the precancerous chronic condition may be the cause of the altered metabolism which produces the growth stimulating substances. In other papers I have given reasons for regarding the overcrowding of the lymphocytes as an essential step towards malignant disease, and the presence of microorganisms, especially the tubercle bacillus, as the factor which brings about the overcrowding of these cells.

Cancer of the Stomach.

Occurring only as a rare curiosity in any of the lower animals, cancer of the stomach presents the most common tumour in human beings. In England the incidence in men is greater than in women, the proportion being 100 to 83, but this relation in regard to the sexes is not the same in all parts of the world. This disease is appreciably more common in rural districts than in cities, and this peculiarity has been noted in several European countries. Thus the proportion of the total deaths from cancer in men contributed by the stomach is 39·8% in Switzerland and 51% in Baden.

The Decennial Supplement of the Registrar-General contains the age distribution of deaths from cancer in the male population aged sixteen and upwards for the three years 1921–1923. Information is given separately for the following sites: skin, lip, tongue, œsophagus and stomach. Standardized rates for each site are given for the age periods 20–65, but as nearly 45% of the deaths from cancer fall after 65, I have adopted the plan of taking the proportional incidence of the stomach as a percentage of the total deaths from cancer in each occupation. This difference in the method of calculation has not altered the main position of any occupation as high or low, but it may alter the position of each of a group of occupations with a nearly equal incidence of cancer.

The proportion contributed by the stomach to the total deaths from cancer varies greatly in different occupations, ranging from 6% to 50%, the average for all the occupations being 22%. Light on the causation of the disease appears to come from the occupations with the lowest and highest incidences, and I have omitted from the following lists about two-thirds of the total deaths in which the incidence lies between 16% and 25%. The major and some of the smaller occupations are grouped solely by the proportional incidence of cancer of the stomach, without reference to the rate for cancer at all sites.

A. Proportional incidence very low; phthisis low: Barristers, solicitors, architects, civil engineers, Anglican and Roman Catholic clergy, medical men, music teachers, brewers and auctioneers.

B. Proportional incidence very low; phthisis moderate: Railway, bank and civil service clerks, dentists, artists, musicians, domestic servants (male), wool sorters, cotton tenters, makers of leather goods, cellarmen, chimney sweeps.

C. Incidence 25%: Railway porters, wool factory hands, cereal and provision merchants, plasterers, tobacco factory hands, navvies, riveters.

D, E, F, G. Incidence 26% and upwards.

D. Farmers and farm workmen.

E. Coal miners.

F. Tin and copper miners, stone and slate quarriers and masons, metal grinders, cotton blow room hands, rag grinders, cotton weavers, wool tenters, foremen wood workers, foremen building trades, grain millers, paper factory hands, engine-drivers (not railway).

G. Insurance officials and agents, dissenting clergy, railway signalmen, motor drivers, theatre and picture managers, shipyard labourers, canvassers and van salesmen.

The sequence of the occupations in this list at once suggests: (*a*) the negative evidence in regard to the influence of education, (*b*) positive evidence in regard to slate and other irritant dusts, (*c*) a predilection on the part of certain rural occupations, especially farmers and their men.

The Influence of Education.

The occupations in Groups A and B may be divided into an educated section including 12,000 deaths, mostly those of persons of professional standing, and a section including 8,000 deaths comprising clerks (railway, bank and civil service), male domestic servants, chimney sweeps, brewers, cellarmen and three smaller trades. In both sections the stomach is affected in about one-seventh of the deaths from cancer, but the first section shows 12·8% of the total deaths as due to cancer and 5·4% to phthisis, while in the second section the incidence is cancer 15% and phthisis 14%. The questions are: is the difference between 15 and 5·4 in the incidence of phthisis a factor in the lower rate for cancer, and to what extent is this difference due to education in the broader meaning of the term.

At the last annual meeting of the Society for the Prevention of Tuberculosis, Professor Sir J. Robertson, of Birmingham, showed that the mortality from all forms of that disease was nearly three times as great among unskilled workers (Social Class V) as among the middle-classes, and that it was nearly twice as great among skilled workers (Social Class III) as among Social Class I.[10] In other words, the risk of the spread of the disease is at a minimum among those who know the facts and are able to control their surroundings, and in this sense education has enabled us to control tuberculosis. A survey of the occupations in these groups makes it clear that the lowest incidence of tuberculosis and of cancer of the stomach are associated with precisely the same conditions of life.

The Influence of Rural Life.

The occupations grouped under D and G include 45,000 deaths, mainly of farmers and farm workmen, together with smaller numbers from railway signalers, insurance agents, motor drivers and a section of the clergy. The incidence of cancer is 12·3% and of phthisis 4·4%. Hence in both diseases they give somewhat better results than the educated classes, except that 27% of the cancer affects the stomach, instead of less than half that ratio. Why do these people living in rural districts present the best results so far attainable in England in regard to both diseases, and yet the stomach is affected twice as frequently as the average of the occupations in Group A? Does this suggest something associated with the farm as the vehicle of the special irritant?

Coal Mining.

Coal miners form a special section represented by 27,500 deaths, in which cancer and phthisis each account for about 10%, the stomach being the site in nearly one-third of the cases of cancer. Coal miners have always been noted for a low incidence of tuberculosis as compared with other miners, and this peculiarity has been observed in many parts of the world. It would almost appear that the lungs are in some way protected by the coal dust from the risk which is at a maximum in the dust of siliceous rocks. Among the underground workers the "hewers and getters" have the highest proportional incidence of cancer of the stomach, and this varies in the different fields, the lowest being 20% in Gloucester and Somerset, and the highest 63% in Cumberland. But as the other underground workers as well as the surface men all present abnormally high rates for the stomach, the agent would appear to be associated with the coal rather than with the varying character of the rock in which the seams are embedded. In other words, it is possible that coal dust taken with the food may have some action on the stomach, even if it is comparatively harmless when inhaled into the lungs.

Dust as a Definite Irritant.

Group F is represented by 22,300 deaths, with 11·4% cancer and 12·7% phthisis. Rather more than every third case of cancer affects the stomach. More than two-thirds of the persons in these occupations are exposed to the risks of a dusty atmosphere, and they include some of those with the highest incidence of phthisis. The character of the dust varies, as may be seen from the arrangement of the occupations in the list. The miners and men working in stone are followed by the textile trades, and these by wood dust, flour and coal smoke. The last three of these appear to produce no injury to the lungs. On the other hand, quarrymen and workers in slate merit special attention, for they have the highest incidence of cancer of the stomach, and they have been celebrated for the prevalence of both phthisis and cancer for more than forty years. The slate quarries are chiefly situated in the rural district of Gwyrfai, Carnarvonshire, the records of which in 1930 were: Total deaths (less accidents), males 170, females 203; phthisis, males 22, females 25; cancer, males 23, females 33.[11]

Figures such as these have led to repeated investigations, so that the facts may be accepted as well established. There is a larger excess mortality from phthisis for workers than for quarriers. The latter are not subjected to much slate dust, while the men in the sheds are constantly inhaling it. The phthisis proportional incidence is workers, 21%; quarriers, 12·5%; cancer, all sites, workers, 11%; quarriers, 9·7%; of which the stomach contributes 50% in workers and 42% in quarriers. The slate dust therefore seems to make comparatively little difference to the incidence of cancer as a whole, however much it may affect the lungs. But in both classes the workmen would have ample opportunity of conveying slate dust to their food. As in the case of slate workers, tin and copper miners also have long had a bad reputation for both phthisis and cancer, and here again about one-third of the cancers affect the stomach.

Finally, there are 7,500 deaths in Group G which occur in occupations which appear to be as hygienic as the average. Insurance officials and agents appear to be affiliated in other respects to the professional class. It may, however, be noticed that theatre managers and proprietors and motor drivers are two recently developed occupations, and in consequence the older age groups are very small as compared with those earlier than 55. Hence as cancer of the stomach attains its maximum proportional incidence in the age group 35–45, it may be that in such occupations it will assume a more normal position in future census periods.

The inclusion of a section of the clergy in this group is probably an accident, because it happens that in one age group nine of the eighteen deaths from cancer affect the stomach—a site distribution perhaps abnormal.

Food Conditions Suspected.

In reviewing the facts thus assembled, it should be mentioned that the point of view taken by the medical staff of the Registrar-General's Department is that cancer of the stomach is in some way dependent on the food. They regard two facts as very significant—first the regularity of the increase in the incidence for the sites in the upper part of the alimentary canal (lip, tongue, mouth, œsophagus and stomach) as well as for the skin, from a minimum in Social Class I to a maximum in Class V. But this uniformity ceases at the pylorus, and there is little difference among the social classes in the incidence of cancer of the intestines, liver, pancreas, bladder, prostate, lung, bones and lymphatic glands. In the second place, as the skin, lip, tongue and mouth usually proliferate in response to definite irritants reaching them by way of the surface epithelium, it is reasonable to suppose that some similar factor influences the stomach also, but does not pass beyond the pylorus. Unless the stomach is affected by a factor very similar to that operating on the other sites of the upper alimentary canal, it is difficult to see why the regularly graded incidence found in all the five social classes should not apply to sites beyond the pylorus also.[12]

The article of food or drink in question does not appear to be alcohol, because no correlation can be

established between cirrhosis of the liver (which is taken as the criterion of alcoholism) and cancer of the stomach. The proportional site incidence of the stomach is very low in brewers and cellarmen, and medium in publicans, barmen and waiters. In this respect the stomach differs from the tongue and œsophagus, both these sites having correlations with alcohol, and to a less degree with syphilis. Recently, attention has been drawn to the association of cancer of the œsophagus and alcohol in foreign countries also.

The only irritant disclosed in my analysis of the incidence of the disease is dust, and this can be identified so definitely in the larger occupations of Group F that its influence can be safely extended to smaller bodies of workers also. If this evidence referred to a disease of the lungs, the evil effect of the dust would at once be conceded, as is regularly done in miners' silicosis. Parity of reasoning from the lung combined with the foregoing line of evidence worked out by the Registrar-General makes it very probable that the gastric irritant in these occupations is associated with dust.

The insolubility of the particles of dust seems to be an important point, for lime and cement workers and limestone quarriers and masons are occupations with a low incidence, whereas the insoluble particles are so varied in character in those with a high incidence that it is possible that they act merely as vehicles for the active agent. This applies to silica, slate, coal, timber, tobacco, cotton and wool. It will also be noted in regard to these dusts that they are all, except coal, associated with a high incidence of tuberculosis, and in the rural district inhabited by the slate workers the women suffer more heavily than the men, both from tuberculosis and from the total incidence of cancer.

Tuberculosis and Cancer.

But the evidence from the forty thousand deaths of farmers and their men seems decisive against dust *per se* being the active agent, for it is absent in this group, and also from a number of other occupations which have a high incidence of cancer of the stomach. The product of the farm which may explain such peculiar instances and at the same time coordinate all our observations is milk, for milk in England is very frequently infected with the tubercle bacillus. Now while milk is known to produce tuberculosis in children, infection of adults appears to be a very rare event. Hence it has come about that the danger from bovine tubercle bacilli by way of the intestinal tract is looked upon as negligible. Nothing is known regarding the ultimate fate of the organisms, but because they have rarely been recovered from enlarged lymphatic glands in the adult, it is presumed that they disappear without leaving any trace of their presence. As practically every pint of milk is consumed in one form or another from day to day, it means that the whole community comes in contact with the bacilli. My own experiments show that ulcers of the stomach, some of which are malignant, may follow the inoculation of mice with tubercle bacilli. It is therefore suggestive to find that the two very large occupations which have most to do with milk are also those which exhibit a high incidence of cancer of the stomach, and that the other occupations which hold a similar position in regard to this disease have probably an average consumption of milk, but the ingestion of dust of many varieties seems to act as an adjuvant. Just as in the case of the lungs, a dusty atmosphere *plus* the tubercle bacillus gives rise to the most formidable of all occupational risks, so the presence of dust in one section of the occupations and of food containing tubercle bacilli in another section, both of which, however, agree in having a very high proportional incidence of cancer of the stomach, is sufficient to raise the question of cause and effect. Evidence as to the close association of phthisis and cancer is found in the comments of the Registrar-General on several of the 178 occupations. He notes that there is an unduly high incidence of both these diseases in the following:

Metal grinders	Textile warehousemen
Cutlers	Musicians
Iron miners	Bus and tram conductors
Pottery furnace men	Carriers and drivers of horse vehicles
Skilled glass workers	Shipyard labourers
Brass finishers and turners	Dock labourers
Rag grinders	Chimney sweeps
Hatters	Cellarmen
French polishers	Barmen and waiters.
Upholsterers	

He also draws attention to an apparent anomaly in certain occupations. In woodworking foremen "the record of outstanding mortality from cancer of the stomach is somewhat surprising, as the rate for all sites is low; but the number of men involved is large, and the result is therefore significant". The general mortality rate for grain millers is low; there are only nine occupations with a lower rate for cancer, yet 28·6% of this rate is due to cancer of the stomach. Metal polishers share the special excess of mortality from respiratory diseases and cancer first noted for the two chief silica risks—tin and copper mining and metal grinding. Cotton blow room hands are engaged on the preliminary preparation of the fibre, and are exposed to much dust.

All the occupations specially exposed to dust are included in the foregoing groups with a high incidence of cancer of the stomach, with the exception of sweeps and cotton spinners. In these two cancer of the skin is extremely common and bears the brunt of the disease. The intermediate Group C, with 25% affecting the stomach, is a stepping stone to the groups with the higher incidence, because four of its seven occupations are subject to dust. If foodstuffs are of special significance, one at least of the occupations in Group G may be classified as milk drinkers.

The occupations in Groups E and F exhibit a very high incidence associated with average conditions of exposure to tubercle bacilli and dusty conditions of the walls of the stomach. Those in Group D exhibit high incidence, associated with average conditions of the interior of the stomach and excessive exposure to tubercle bacilli. The slate workers exhibit excess of both conditions associated with the highest proportional incidence of the disease.

The position thus outlined is independent of the truth of the virus theory of cancer, for if it be found that a large proportion of healthy adults harbour the cancer virus, it is clear that this must be set in action by another factor, such as tar, acting on the tissues. The parallelism would be similar to the action of *Bartonella muris* in the rat. This organism appears to be widely present, perhaps in the mucous membrane of the intestines. After splenectomy a percentage of rats develop a severe form of anæmia with Bartonella bodies in the red blood corpuscles. The removal of the spleen enables the organism to invade the tissues. An allied organism is found in the infective wart in man, *Verruga peruviana*; cultures of this organism produce the warts in monkeys. If a virus is the cause of cancer of the stomach, the sequence found in the occupations would suggest that it is set free by an overdose of tubercle bacilli or by the joint action of the bacilli and an insoluble dust. The facts that some anæmias are not far off malignant disease, and infective warts not far removed from epitheliomata, make the above examples of importance to the virus theory. If the virus is found in ordinary epithelial and other cells, but requires another factor to set it in action, we may call these the direct and indirect causes of malignant disease, but it is clear that each is equally necessary to the production of cancer.

The Problem of Tuberculous Milk.

I have on several occasions drawn attention to the facts which may point to the influence of boiling the day's supply of milk in Queensland, a custom more regularly adopted there than in other parts of Australia, on account of the larger proportion of the population living under higher temperatures during daylight in most months of the year. For some reason the death rates for infants from all causes, for tuberculosis in children, for tuberculosis at all ages, and for cancer are lower in Queensland than probably in any other part of the world. The general rate for cancer in both sexes is 90, as against 118 in Victoria, while the proportional incidence of cancer of the stomach in men is 26·5%, as against 30% for the other parts of the Commonwealth. Possibly, therefore, these are other instances of cause and effect.

Without affirming that this survey of the problem of cancer of the stomach is proof that one cause is the consumption of tuberculous milk, I hold that it casts grave suspicion on the bacillus, and the next step should be to incriminate or exonerate that organism. It happens that most civilized people are unwittingly experimenting on themselves by drinking contaminated milk and in that way supplying the conditions under which, if the tubercle bacillus is the cause, cancer would presumably appear, as it does, in people enjoying the best of health. As the stomach is the most frequent site of cancer in both sexes, it is safe to infer from the instances discussed in the occupations that the unknown irritant must be well nigh ubiquitous.

The only way in which the influence of tuberculous milk can be tested is by obtaining volunteers to undertake for a period of at least four years to boil all the milk they consume. Commercial pasteurization is carried out at too low a temperature to fulfil all the conditions of a scientific experiment in testing this question. A group of 20,000 persons above the age of 40 distributed in age groups corresponding to those enumerated at the last census could be obtained from about 8,000 households. Normally this group would have 420 deaths in the first year, including about 70 from cancer. In the second year, if milk is one of the factors in the disease, there would be a slight reduction in the deaths from cancer. The third year would then show a decided reduction, which would become more marked in the fourth and succeeding years.

Summary.

1. A survey of the deaths in the occupations in England shows that in certain sections cancer appears to have reached its maximum.

2. Site incidence depends mainly on the ease with which a definite irritant can reach a suitable tissue.

3. Cancer of the stomach appears to be largely controlled by two factors: (*a*) insoluble dust of animal, vegetable or mineral origin; (*b*) some substance prominent in the environment of farmers and farm workmen.

4. Reasons are given why tuberculous milk thus comes under suspicion, and a scheme is outlined for testing this question.

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PSITTACOSIS IN AUSTRALIAN PARROTS.[1]

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PROBABLY on account of the absence of recorded human cases, it has been generally assumed that psittacosis is not present amongst Australian parrots. Meyer and Eddie,[1] however, recently reported that budgerigars caught in the wild state near Adelaide and shipped at once to California, were found to be infected with a weakly virulent strain of psittacosis. Information has also been received that two cargoes of parrots consigned from Australian ports to London early in this year suffered a high mortality on the voyage, and on arrival were found to be infected with typical psittacosis virus.

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In addition to these recent reports, Merrilees[2] has described an infection occurring in 1930 amongst caged budgerigars in Melbourne. Though the findings reported are not quite conclusive, it seems probable that this epizootic and an associated human infection were both due to psittacosis virus.

The present investigations were initiated at the request of the Commonwealth Director General of Health, in order to determine definitely whether psittacosis was endemic amongst parrots in this country. This preliminary report is concerned merely to establish the fact that a considerable proportion of parrots purchased from dealers in both Adelaide and Melbourne are infected with typical strains of psittacosis virus.

Birds Received from Adelaide.

A crate containing twenty-four parrots was received from an Adelaide dealer on September 13, 1934. The following species were present in the consignment: Grass parrakeet or red-backed parrot (*Psephotus hæmatonotus*), eleven individuals; rosella (*Platycercus eximius*), five individuals; Adelaide rosella (*Platycercus adelaidæ*), four individuals; cockatiel (*Leptolophus hollandicus*), four individuals.

None appeared obviously sick, but no examination of individual birds was made before they were killed. The crate was covered with a cloth wet with cyllin antiseptic, and coal gas was run in to kill the birds. The whole contents of the cage were thoroughly wetted with cyllin, and the parrots were removed.

Post mortem examinations were made immediately with full aseptic precautions. Particular attention was paid to the spleen, which was the only organ in which any abnormalities were noted. The organ was removed aseptically from all birds, and its diameter was noted. (All spleens were approximately spherical.) These diameters are shown in Table I in millimetres.

TABLE I.

Red-backed parrots—	
Males	5·5, 3·0, 4·0, 3·0, **8·5**, 4·4, **9·0** (Numbers 10–16).
Females	3·5, 3·5, 5·0, 4·5 (Numbers 1–4).
Rosellas	5·5, **10·0**, 4·5, 3·8, **10·0** (Numbers 5–9).
Adelaide rosellas	3·0, 4·0, 4·0, 3·5 (Numbers 17–20).
Cockatiels	**8·0**, **7·0**, **8·0**, **9·0** (Numbers 21–24).

Spleens from parrots Numbers 6, 9, 14 and 16 were obviously abnormal, being much larger and more engorged than normal; Number 16 was very dark, and looked almost necrotic. All four cockatiels had large spleens not obviously different from, for example, Number 6 or Number 9, but in the absence of any other birds of this species, it cannot be proved that these were abnormally large for the species.

Emulsions in broth were made from the following spleens individually: Numbers 5, 6, 9, 10, 14, 16; and from the following "pools": Numbers 1, 2, 3 and 4; 7, 8; 11, 12, 13, 15; 17, 18, 19, 20; 21, 22, 23, 24. The spleens were ground with quartz powder and about four cubic centimetres of broth to each batch. The emulsion was tested for sterility by removal of a few drops of broth and was used for the inoculation of mice (0·5 cubic centimetre intraperitoneally) and developing hen eggs (0·03 cubic centimetre) on to the chorio-allantoic membrane. In addition, portion of the emulsions 6 and 9 were pooled, filtered through a sand and paper pulp filter with a little extra broth, and then through a permeable Elford membrane capable of retaining bacteria. This filtrate is termed MF6+9 in the table. All emulsions and filtrates were found to be sterile with the exception of spleen emulsion 9, which contained a few coliform bacteria and was discarded. The mice were all placed in individual glass jars and examined for signs of illness (ruffling of fur and inactivity) daily. The results of mice inoculations are shown in Table II.

TABLE II.

Pathogenicity of spleen emulsions and filtrates for mice by intraperitoneal inoculation.

Inoculum.	Mouse Number.	
MF6+9	47	No illness. Killed tenth day. Normal.
	48	
Spleens 1–4	59	No illness. Killed tenth day. Normal.
	60	
Spleen 5	53	No illness. Killed tenth day. Normal.
	54	
Spleen 6	49	Sick fourth day. Killed tenth day. Spleen ++, peritonitis.
	50	Sick fourth day. Killed fourth day. Spleen and liver +. No Levinthal-Coles-Lillie bodies seen.
Spleens 7 and 8	61	No illness. Killed tenth day. Normal.
	62	+ third day.
Spleen 10	63	+ third day.
	64	No illness. Killed tenth day. Normal.
Spleens 11, 12, 13, 15	65	No illness. Killed tenth day. Normal.
	66	
Spleen 14	55	Sick fourth day. Killed fifth day. Spleen and liver +. Levinthal-Coles-Lillie +.
	56	Sick fourth day. Killed seventh day. Spleen +. No Levinthal-Coles-Lillie bodies seen.
Spleen 16	57	Sick fourth day. Killed sixth day. Spleen and liver +. Levinthal-Coles-Lillie +.
	58	Sick fourth day. Killed fifth day. Spleen and liver +. Levinthal-Coles-Lillie +.
Spleens 17–20	67	No illness. Killed tenth day. Normal.
	68	
Spleens 21 and 20	51	No illness. Killed tenth day. Both spleens +.
	52	52 some peritonitis.
Spleens 23 and 24	69	No illness. Killed tenth day. Spleens slightly +,
	70	but otherwise normal.
	Intracerebral inoculations—	
MF6+9	Three mice. All sick on fourth day; one dead and others killed for passage on fifth day.	
Spleen 16	One mouse. Moribund and killed on fourth day.	
Spleens 23 and 24	Sick on tenth day, and killed for passage.	

These afford presumptive evidence that psittacosis virus was present in parrots 6, 14 and 16, and possibly in both cockatiel "pools" 21–22, 23–24. The filtrate from spleens 6+9 gave no evidence of infection by the intraperitoneal route, but was obviously infective when given intracerebrally. Primary egg inoculations with the same materials gave lesions which are now known to be characteristic of the virus under discussion with MF6+9 and spleen 16 emulsion. Passage of the virus strains has been readily accomplished, and may be briefly recorded according to the species from which they were derived.

Rosella Strain.—Starting with the membrane filtrate 6+9, definite lesions were present after intracerebral inoculation, and in both hen and duck eggs. Intraperitoneal inoculation of brain from first generation mouse gave typical lesions. In connexion with other experimental work, this strain has been grown for five generations in developing duck eggs, giving numerous typical Levinthal-Coles-Lillie bodies in smears from the chorio-allantoic membranes.

Grass Parrakeet Strain.—Using spleen 16 as starting point, peritoneal, intracerebral and egg transfers have all been readily accomplished. A membrane filtrate, made from liver and spleen of a second generation mouse, was infective by intracerebral inoculation, and subinoculation intraperitoneally from this brain gave a typical lesion with Levinthal-Coles-Lillie bodies. This is the most virulent strain for mice that has so far been encountered.

Cockatiel Strain.—Intraperitoneal inoculations gave dubious results, and intracerebral inoculation only resulted in sickness on the tenth day. Subinoculation from this brain, however, was definitely positive, one mouse dying and the other being killed when moribund on the sixth day. Third generation inoculations gave typical lesions with Levinthal-Coles-Lillie bodies in mice intraperitoneally, and the characteristic lesions on egg membranes.

Summary.—Virus strains were obtained from rosellas and grass parrakeets with enlarged spleens which showed all the characteristics of psittacosis virus, namely, inability to grow in ordinary media, pathogenicity for mice, both intracerebrally and intraperitoneally, filtrability and power to produce the characteristic Levinthal-Coles-Lillie bodies in infected cells. The cockatiels also provided a similar strain not yet proved filtrable, and of lower virulence, but of obviously similar nature. It is reasonable to conclude that all the birds in this consignment showing spleens seven millimetres or more in diameter were carriers of psittacosis, that is, 33%.

Parrots from Melbourne Dealers.

First Melbourne Group of Birds.

Twelve grass parrakeets (*Psephotus hæmatonotus*) were purchased from Melbourne dealer "A" and were killed and examined as before. A large proportion of these showed splenic enlargement, and many had, in addition, chronic or subacute thickenings in the walls of the abdominal air sacs and in the peritoneum, especially around the spleen. Spleen diameters and other lesions were as shown in Table III.

TABLE III.

Parrot.	M1	M2	M3	M4	M5	M6	M7	M8	M9	M10	M11	M12
Spleen diameter in millimetres	9·5	7·5	7·0	7·0	6·0	3·0	8·0	8·0	9·0	8·0	9·0	4·0
Air sac thickening ..	−	+	+	−	−	−	+	+	+	+	+	±

Tests for sterility showed only spleen M8 to be contaminated with bacteria. Four large spleens, M1, M3, M7, M9, were chosen for investigation, and were ground separately with broth and powdered quartz and inoculated into mice and eggs. In this series each emulsion was inoculated into one mouse intracerebrally (0·02 cubic centimetre), one intraperitoneally, and on to the chorio-allantoic membranes of two eggs. The results were as shown in Table IV.

Levinthal-Coles-Lillie bodies were found only in material from mice and eggs inoculated from M9, but the general resemblance amongst the results of

TABLE IV.

Pathogenicity of spleen emulsions from Melbourne Parrakeets.

Parrot.	Mouse Intracerebral Inoculation.	Mouse Intraperitoneal Inoculation.	Egg Inoculation.	
M1	Sick; killed in five days.	Killed on eighth day. Typical peritonitis and splenic enlargement.	+	±
M3	Sick; killed in four days.	Killed on eighth day. Typical peritonitis and splenic enlargement.	±	±
M7	Sick; killed in four days.	Killed on eighth day. Typical peritonitis and splenic enlargement.	+	+
M9	+ five days.	Killed on eighth day. Typical peritonitis and splenic enlargement.	+	+

Egg inoculations, + = typical lesion; ± = a few foci resembling those obtained with active material.

the inoculations, as well as the almost uniform anatomical findings in the parrots, makes it reasonably certain that all four were infected with a single strain of psittacosis virus. Up to the present, further passages have been made only with material from M9. The primary egg lesion was emulsified in a few cubic centimetres of broth, and the supernatant broth was titrated by intracerebral inoculation in mice. It was active at a dilution of 10^5. Passage from the mouse liver to eggs has also been made with this strain, and numerous Levinthal-Coles-Lillie bodies have been observed in egg membrane smears.

Summary.—Of these twelve parrots nine or ten were probably carriers of psittacosis.

Second Melbourne Group of Birds.

From a second Melbourne dealer "B" eighteen rosellas and six crimson parrots (*Platycercus elegans*) have been obtained and examined. Fourteen of these showed definitely enlarged spleens over six millimetres in diameter, and three had, in addition, chronic or subacute thickenings of air sac membranes. Investigations on this group are still in progress, but one active strain of psittacosis virus has been definitely established. Several of the enlarged spleens appear to contain strains of low virulence for the mouse, and passage experiments with these are being made. In all probability these splenic enlargements are all, or nearly all, indicative of infection with psittacosis.

Discussion.

The virus strains which have been obtained from these Australian parrots present all the classical characteristics of psittacosis virus. They vary amongst themselves in virulence for mice, but on the whole appear to be less virulent than the strains isolated from and responsible for the human cases in the European and American outbreaks of 1929–1930.

There is no evidence yet available to allow any statement in regard to the possible danger of human infection from what is apparently an extensive "reservoir" of virus in parrots. It is possible that the strains concerned are non-virulent for man, but it would seem more likely that they are occasionally responsible for human infections.

In the circumstances it is important that any cases of suspicious infections in persons in contact with parrots should be adequately investigated. Typical

psittacosis in man is a serious prolonged illness with a high mortality, but it is almost certain that infections of every degree of severity can occur. Influenza-like infections occurring outside epidemic periods, and particularly those with broncho-pneumonic signs, should be regarded with suspicion. Diagnosis can only be established with certainty by the isolation of the virus from the sputum.

Summary.

Psittacosis virus has been isolated from parrots obtained from dealers in Adelaide and Melbourne.

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OBSERVATIONS ON THE GUANIDINE, GLUCOSE AND CALCIUM CONTENT IN THE BLOOD IN ECLAMPSIA.

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IN 1928 Minot and Cutler[1] drew attention to the similarity in symptoms produced in dogs by carbon tetrachloride, chloroform or guanidine intoxication, and also to the variations in certain chemical constituents of the blood. An appreciable rise in guanidine concentration, associated with a fall in blood sugar and in calcium, was observed in each of these conditions. The blood sugar could be restored to its normal level, and the clinical state definitely improved, by giving calcium salts, such as the gluconate or the lactate, whereas with administration of glucose the blood sugar returned to its normal level, but the symptoms of poisoning remained.[1] The outstanding pathological changes produced in these animals were severe central necrosis of the liver. This fact led to the assumption that the increase in guanidine was related to disturbed metabolism due to injury of the liver.

A comparison was then made between the symptoms in eclampsia and those in cases of intoxication produced in dogs by the abovementioned drugs.[2] The clinical findings in both human beings and dogs were characterized by more or less acute liver injury, gastro-intestinal irritation, nervous disturbances, and tendency to hypoglycæmia. A small series of eclamptic and preeclamptic patients were investigated by these authors.[2] They observed that the fasting blood sugar level decreased in preeclampsia to 60 to 70 milligrammes per 100 cubic centimetres of blood, whilst in eclampsia the hypoglycæmia was still more pronounced.

In normal pregnancies the guanidine in the blood ranged from 0·25 to 0·40 milligramme per 100 cubic centimetres of blood, whilst in eclamptic and preeclamptic patients a higher level was found, ranging from 0·5 to 0·85 milligramme per 100 cubic cubic centimetres of blood.

Recently Bartholomew and Parker[3] suggested that guanidine was the toxin responsible for the production of eclamptic and preeclamptic toxæmias, since this substance has been shown to increase the excitability of motor nerve endings to constant electric currents. In eclampsia and preeclampsia the muscles are rendered hypersensitive, as shown by the frequent twitchings and the ease with which convulsions are precipitated by sudden noises or trivial disturbances. Harding and Fort[4] have shown that placental tissue contains twice as much argenine as is present in other tissues. Normally, argenine is converted to urea and ornithine by the action of arginase. Bartholomew and Parker[3] indicated a process of deaminization, oxidation and reduction whereby argenine might be converted to guanidine. They suggested the possibility of argenine being produced as the result of autolysis of placental infarcts and the decomposition of this argenine liberating sufficient guanidine to cause convulsive seizures. The action of guanidine may be modified by the number, size and location of the infarcts, by the degree of blockage of the circulation, and by the rate of autolysis.

In 1932 Stander[5] reported the guanidine base concentration in the blood in a series of eclamptic patients, and made the following statement:

> We must conclude that our patients did not show a definite or marked increase in guanidine, nor a hypoglycæmia, and in this respect our results are at variance with those of Minot and Cutler.

Stander's series consisted of five normal patients and twenty-three eclamptics. His results for guanidine are summarized in Table I.

TABLE I.
Guanidine content in blood, expressed in milligrammes per 100 cubic centimetres of blood.

Type of Patient.	Minimum.	Maximum.	Average.
Normal	0·22	0·40	0·33
Eclampsia	0·26	0·58	0·39

In this series the guanidine concentration in the blood exceeded the value 0·40 milligramme in only three cases.

Since this work by Stander was published, several papers in which guanidine was regarded as the toxic factor responsible for the eclamptic toxæmia have appeared. That of Bartholomew and Parker[3] appeared as late as 1934.

Analyses of the blood from eclamptic patients have been performed in this laboratory since 1931, and included guanidine, calcium, sugar and urea estimations. The results which have been obtained confirm the findings of Stander with regard to guanidine, and make the theory that this substance is the toxic factor responsible for eclampsia seem improbable.

The blood was obtained from eclamptic patients as soon as possible after admission to hospital and before any treatment had been commenced. The methods used in these estimations were: (i) guanidine, Major and Weber[9]; (ii) glucose, Maclean[10]; (iii) urea, Maclean[11]; (iv) calcium, Kramer and Tisdall-Clark and Collip.[12] The detailed results are shown in Table II.

TABLE II.

Correlation of Chemical Figures and Clinical Data in a Series of Thirty Cases of Eclampsia.

Case Number.	Guanidine. Milligrammes per 100 Cubic Centimetres of Blood.	Blood Sugar. Percentage.	Calcium. Milligrammes per 100 Cubic Centimetres of Blood.	Blood Urea. Milligrammes per 100 Cubic Centimetres of Blood.	Fouchet.	Blood Pressure.	Albumin.	Number of Fits.	Toxæmia.	Remarks.
I	0·21	0·144	9·6	26	No reaction.	—	—	—	Eclampsia.	
II	0·19	—	—	—	—	150	¼ solid (rapidly cleared).	7	*Ante partum* eclampsia.	On admission comatose, œdema of face and ankles. Responded to treatment.
III	0·20	—	—	—	—	150	¼ solid (cleared slowly).	5	*Ante partum* eclampsia.	Fairly ill patient.
IV	0·21	—	—	—	—	140 (rapidly fell).	¼ solid.	—	Eclampsia.	Œdema, headache, vomiting.
V	0·24	0·134	8·8	35	No reaction	130	Solid.	—	*Ante partum* eclampsia.	Induction of labour with rectal tubes eight days after admission.
VI	0·26	0·100	10·5	13	—	125	¼ solid.	4	*Intra partum* eclampsia.	Severe headache, vomiting.
VII	0·28	0·087	8·8	15	No reaction.	118	¼ solid (cleared rapidly).	8	*Ante partum* eclampsia and pyelitis.	Œdema and headache for four days before admission.
VIII	0·28	0·139	10·3	10	Positive.	—	—	—	Eclampsia.	
IX	0·30	—	—	—	—	160	Solid.	7	*Ante partum* eclampsia.	Albuminuria, headache, giddiness for six days prior to admission.
X	0·32	0·091	8·6	58	Positive.	—	Solid.	7	Eclampsia and chronic nephritis.	Cæsarean section, collapse and death. *Post mortem:* Bilateral renal calculi, chronic nephritis and cardiac failure.
XI	0·32	0·125	8·8	42	Positive.	195	Solid.	6	Eclampsia and chronic nephritis.	Abdominal hysterectomy and sterilization.
XII	0·32	0·072	9·4	39	No reaction.	180	¼ solid.	1	*Intra partum* eclampsia.	Albumin noted in the urine for two weeks. Semi-conscious on admission.
XIII	0·33	0·079	10·7	16	No reaction.	160	¼ solid.	8	*Ante partum* eclampsia.	Severe headache and oliguria. Induction of labour with rectal tube on day of admission. Condition gradually improved.
XIV	0·33	—	8·0	28	No reaction.	—	—	—	Preeclampsia.	
XV	0·33	0·143	9·0	40	Positive.	—	—	—	—	
XVI	0·34 —	0·134 0·065	8·7 8·7	18 19	No reaction. No reaction.	165	Solid (persisted).	8	*Intra partum*.	Oliguria. Very ill woman.
XVII	0·34	0·095	8·1	22	No reaction.	210	Solid.	1	*Intra partum*.	Very sick patient, comatose, restless, irritable, blurring of both disks. Death fifteen hours after delivery.
XVIII	0·35	—	9·9	30	—	160	¼ solid.	—	Eclampsia and chronic nephritis.	Stone removed from bladder and acute nephritis, three years previously. Albuminuria for four weeks before admission.
XIX	0·36	—	—	34	—	168	Solid.	3	*Intra partum* eclampsia.	On admission semi-conscious and irritable. No response to treatment. Death on day of admission.
XX	0·41	0·074	8·7	—	No reaction.	170	Solid	3	*Ante partum* eclampsia.	History of headache, vomiting, œdema for three days. On admission "blind", extreme headache, oliguria, vomiting, semi-conscious. Induction of labour with rectal tube. Albuminuria rapidly subsided, and blood pressure returned to normal.

TABLE II.—*Continued.*

Correlation of Chemical Figures and Clinical Data in a Series of Thirty Cases of Eclampsia.—Continued.

Case Number.	Guanidine. Milligrammes per 100 Cubic Centimetres of Blood.	Blood Sugar. Percentage.	Calcium. Milligrammes per 100 Cubic Centimetres of Blood.	Blood Urea Milligrammes per 100 Cubic Centimetres of Blood.	Fouchet.	Blood Pressure.	Albumin.	Number of Fits.	Toxæmia.	Remarks.
XXI	0·42	0·112	11·8	25	No reaction.	130	¼ solid.	1	*Intra partum* eclampsia.	In coma when admitted.
XXII	0·43	0·164	9·8	16	No reaction.	—	Solid.	2	*Ante partum* eclampsia.	Swelling of limbs for three weeks, headache one week, with vomiting. On admission semi-conscious, spasmodic rigidity, œdema. Death just before delivery.
XXIII	0·43	0·420 (taken after intravenous glucose injection).	8·2	58	No reaction.	180	¼ solid.	3	*Intra partum* eclampsia.	Admitted September 8, 1931, with trace of albumin and blood pressure 166 millimetres of mercury. Cleared rapidly with treatment. Readmitted October 20, 1931, with blurred vision, œdema, blood pressure 140 millimetres of mercury. Readmitted November 3, 1931. Induction of labour November 7, 1931.
XXIV	0·43	0·083	9·0	39	Positive.	—	Solid.	—	Eclampsia.	Severe vomiting, headache, constipation. On admission semi-conscious and restless. Eliminative treatment applied, and her general condition improved. As the blood pressure continued to rise, labour was induced with rectal tube. Blood pressure fell rapidly after delivery.
XXV	0·44	0·083	10·6	31	—	175	Solid.	1	*Ante partum* eclampsia.	Improved slightly with eliminative treatment. Albumin was persistent.
XXVI	0·48	0·074	9·0	—	No reaction.	140	¼ solid.	Several fits.	*Post partum* eclampsia and puerperal pyelitis	Albumin, œdema, polyuria, eye trouble for two weeks. No improvement with treatment, hence labour was induced.
XXVII	0·48	0·080	10·0	27	—	140	Solid.	2	*Intra partum* eclampsia.	Persistent albumin and high blood pressure. Sick woman.
XXVIII	0·51	0·102	9·0	36	—	135	Solid.	8	Eclampsia.	Clinical condition improved after delivery.
XXIX	0·52	0·096	7·0	16	—	160	Solid.	—	Preeclampsia.	Œdema of feet, dimness of vision, headache, breathlessness. Induction of labour with rectal tube.
XXX	0·54	0·196	11·5	34	Positive.	145	Solid.	5	Eclampsia.	Unconscious when admitted, no fits after admission.

The guanidine concentration was determined in the blood from six normal pregnant women, with the following results:

Maximum 0·39 milligramme guanidine per 100 cubic centimetres of blood.
Minimum 0·27 milligramme guanidine per 100 cubic centimetres of blood.
Average 0·34 milligramme guanidine per 100 cubic centimetres of blood.

These figures were in agreement with those of Minot and Cutler, and also of Stander.

In the eclamptic group (thirty cases) the guanidine varied from a minimum of 0·21 milligramme to a maximum of 0·54 milligramme, the average being 0·38 milligramme. Thirteen cases (45%) gave values greater than 0·4 milligramme, the upper limit for guanidine in normal pregnancy, but none of the figures exceeded 0·54 milligramme. These results thus confirmed those of Stander, who showed that no marked rise occurred in the guanidine concentration in the blood in eclampsia. Titus and Willets[6] have shown that if the blood sugar was estimated at frequent intervals during an eclamptic seizure, characteristic variations occurred. Immediately preceding the convulsion the blood sugar showed a

definite fall; as the convulsion commenced, the blood sugar began to rise, and this rise continued after the convulsion had ceased. The blood sugar then dropped to lower levels preceding the next convulsion.

This work was confirmed by Laferty,[7] who regarded the convulsion in eclampsia as a mechanism of the body for combating glucose deficiency.

Stander[8] disagreed with this work of Titus and Willets, and definitely stated that, in his opinion, hypoglycæmia was not the usual accompaniment of eclampsia. As Laferty points out, the blood sugar was not observed at sufficiently close intervals, and thus the periods when hypoglycæmia existed may have been missed. In Table II it will be observed that 33% of the blood sugar values were less than 0·80%, many of the values ranged from 0·80% to 0·11%, and 37% were above this value. In one case the blood sugar was as high as 0·196%.

The blood for these estimations was taken soon after admission of the patient to hospital before any treatment was commenced, and therefore these cannot be classified in relation to the phase of the convulsion. This probably accounted for the range of values observed in these cases. The results certainly seemed to indicate that hypoglycæmia did occur in some cases of eclampsia at some phase of the seizure.

The concentration of calcium in the group of eclamptic cases studied revealed interesting facts. In 68% of cases the value was below the normal level (10 milligrammes per 100 cubic centimetres of blood), while in 39% the value was less than 9 milligrammes per 100 cubic centimetres of blood.

In comparing the figures for individual cases, the contention of Minot and Cutler that high guanidine is accompanied by lowered concentrations of blood sugar and calcium is not verified, as exemplified by Case XXX:

Guanidine	0·54 milligramme per 100 cubic centimetres of blood.
Sugar	0·196 milligramme per 100 cubic centimetres of blood.
Calcium	11·5 milligrammes per 100 cubic centimetres of blood.

and Case XXIX:

Guanidine	0·52 milligrammes per 100 cubic centimetres of blood.
Sugar	0·096 milligramme per 100 cubic centimetres of blood.
Calcium	7·0 milligrammes per 100 cubic centimetres of blood.

In Table III an analysis of the results in twenty cases in which guanidine, glucose and calcium values for each specimen of blood were obtained, disproved the statement that high guanidine was associated with low glucose and low calcium concentrations.

TABLE III.

Analysis of values for Guanidine, Calcium and Glucose in the blood of twenty eclampsia patients.

Case Numbers.	Number of Cases.	Guanidine.	Glucose.	Calcium.
VI XIII	2	Normal	Normal	Normal
X XVII	2	Normal	Normal	Low
XII	1	Normal	Low	Low
V VIII	2	Normal	High	Normal
XI XV X	3	Normal	High	Low
XXI XXV XXVII	3	High	Normal	Normal
XXII XXX	2	High	High	Normal
XXIV XXVIII XXIX	3	High	Normal	Low
XX XXVI	2	High	Low	Low

Normal values: Guanidine, 0·40 milligramme per 100 cubic centimetres of blood; glucose, 0·100%; calcium, 10 milligrammes per 100 cubic centimetres of blood.

Summary.

The guanidine in the blood in a series of thirty cases of eclamptics has been correlated with glucose and calcium concentrations. No appreciable rise in guanidine was observed. In 33% of the specimens of blood examined the blood sugar value was below 0·80%, although only isolated estimations, without reference to the phase of eclamptic seizure, were performed. This indicated the probability that hypoglycæmia did exist at some phase in relation to the convulsion.

In 39% of the eclamptic cases the calcium content of the blood was less than 9 milligrammes per 100 cubic centimetres of blood. The statement of Minot and Cutler that eclampsia was characterized by high guanidine associated with low glucose and low calcium concentration in the blood has thus been disproved.

Acknowledgements.

I wish to thank Sister Richmond and Sister Barkes, of the Women's Hospital, for their help in obtaining the specimens of blood for this investigation.

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SYMPTOM-PRODUCING FACTORS IN RIGHT VISCEROPTOSIS.

By ALAN E. LEE, M.D. (Melbourne), F.R.C.S. (England),
Honorary Surgeon to Out-Patients, Brisbane Hospital.

A STUDY of many hundreds of patients with right visceroptosis (absence of lateral fixation of the ascending colon) has shown that symptom-production in them is limited to certain conditions of posture and muscle tone.

The two positions most uniformly devoid of symptoms are the horizontal and the completely erect posture.

This statement is subject to the limitations that, while the continued maintenance of the prone position is free from symptoms, a minority of patients complain of discomfort while supine, and even more so while lying on the right side. Further, many patients complain of symptoms in the erect posture when physically tired, not appreciating that the lowering of muscle tone so produced has allowed a relaxation from the strictly erect posture to occur by drooping forward of the shoulders and flattening of the normal lumbo-sacral concavity of the back.

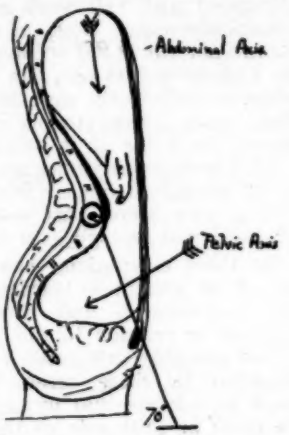
The position above all others productive of symptoms is that of frank forward stooping, especially when associated with physical work. In the standing posture this is produced by such an occupation as loading boxes from the ground on to a lorry, by the use of an ax, and in such agricultural work as raking or hoeing the ground. In the sitting posture the position assumed over a sewing machine and the relaxation associated with lounging in a low canvas chair (such as is found in suburban picture theatres) belong to this type, and in both situations discomfort is very frequently experienced. Many patients have found for themselves that a momentary straightening of the back will suffice to abolish their discomforts till the relaxed position is reassumed.

The only other condition frequently associated with symptom-production is jolting in a sitting posture, such as is associated with horse-riding or tractor-driving, and the jarring effect of walking over rough ground.

The variations from the perfectly erect posture that produce symptoms can be noted to have this in common, that they are associated with a flattening of the normal dorso-lumbar vertebral concavity; that is, they are those positions which decrease lumbar lordosis.

There are two questions that need rational answers before right visceroptosis can obtain general recognition as a pathological entity: First, how can such a widespread condition as non-fixation of the ascending colon (estimated to be partly present in at least 20% of individuals) be held responsible for the symptoms of a much smaller group of patients? Secondly, if such an entity exists, how can the symptom complexes produced by it be recognized?

The answers, as I will endeavour to show, can be deduced from an analysis of the foregoing facts. They are, briefly, that the non-fixed colon produces symptoms only when certain secondary supporting factors fail; and as a corollary, that symptoms produced under the known conditions favouring a failure of such secondary factors are associated in a direct causal relation with right visceroptosis.



Now what are these secondary supporting factors? First, there is the primitive dorsal mesentery, the continuation of the ileal mesentery to the caecum. Secondly, and most important, is the narrowness of the abdomino-pelvic isthmus.

The obliquity of the pelvic brim is normally such that it maintains in the vertical position an angle of 70° with the horizontal. Not only does this cause definite constriction to exist between the two cavities, but also the axes of these two cavities are acutely flexed on each other. A flat abdominal wall, inasmuch as it forms one side of this isthmus, contributes in an important way to its narrowing and *vice versa*.

The descent of the caecum towards the true pelvis also depends on its weight relative to surrounding organs, and here it may be taken for granted that the more full of semi-solid contents the caecum is, the heavier it becomes. This factor of heaviness depends again on efficient colonic function, and usually the degree of constipation becomes the index of this function.

Finally, sudden jolts or jars and the increased abdominal pressure resulting from straining at physical work are factors which help to facilitate colonic descent.

The abdominal and pelvic cavities are pivoted on each other at the lumbo-sacral articulation, increase of their angulation to each other increasing the dorso-lumbar concavity of the back (producing lordosis), while decrease of their angulation decreases or undoes lordosis.

Factors, then, that widen and straighten the abdomino-pelvic isthmus are: (i) a decrease in lumbar lordosis, (ii) lack of tone in the anterior

abdominal wall, (iii) stooping, which tends to convert the abdominal and pelvic cavities into one straight tube.

Now we have already seen that these are exactly the postural factors that favour symptom-production in right visceroptosis. It can then be stated with a considerable degree of certainty: (i) that a right visceroptotic gets symptoms when the caecum prolapses towards the true pelvis; (ii) that such prolapse occurs only when certain secondary factors of posture and muscle tone are brought into action; (iii) that the secondary factors act by: (*a*) widening and reducing the angulation of the abdomino-pelvic isthmus, (*b*) increasing the weight of the caecum, (*c*) facilitating its descent by jarring or jolting, or by increasing intraabdominal pressure.

The treatment of a right visceroptotic comprises two stages: (i) By increasing the efficiency of the secondary supporting factors he can be brought back into the great group of symptom-free visceroptotics; or (ii) by adequate fixing of the ascending colon he can be removed from the group altogether.

Stage (i) is usually the only form of treatment necessary. By attention to posture, improvement of muscle tone and increasing colonic efficiency, the majority of patients can be made fairly comfortable. Some discomforts will usually remain, but if the physical condition present is carefully explained to the patient, if he understands that nature has dealt unkindly with him, and that he cannot expect the completely symptom-free abdomen of the non-visceroptotic, and if finally he acquiesces in this state of affairs, it is rarely that any more radical treatment will be demanded.

Stage (ii) is needed by a restricted group of persons, usually men whose work entails considerable stooping. No improvement in the secondary supporting factors can keep these men symptom-free, and it is important to fix their colons before the oft-repeated discomfort builds up a superimposed neurosis.

An occasional person who takes unkindly to the necessary discipline of Stage (i) had better also be treated by operation.

The improvements in posture and muscle tone can be attained by the simplest forms of physical therapy. It is important that the masseur should understand the type of posture to be striven for. In so far as this demands an increase in lumbar lordosis, it runs counter to certain orthopædic principles strongly ingrained in most physical therapy departments.

Goldthwaite and the Boston school of orthopædists teach that in a somewhat similar group of patients (clinically) to those discussed in this paper postural defects are a common ætiological factor, and they emphasize the undoing of lordosis as one of the primary aims of treatment.

If the right visceroptotic group is treated on these lines it can only result in further weakening of the already insufficient factors, which have been shown to control symptom-production in these persons.

Reports of Cases.

EXTREME CYANOSIS OF DOUBTFUL ÆTIOLOGY.[1]

By R. WHISHAW, M.B., Ch.M. (Sydney),
Hobart.

MRS. M., who has kindly come here for demonstration, presents a case full of interest from the point of view of diagnosis. I shall give you first a brief outline of her history and then discuss, also briefly, my views on the conditions found. As you see, she is now in fair health.

Apart from a tremendous gain in weight, from 75·6 kilograms to 126 kilograms (twelve to twenty stone), over a period of about two years, the patient's previous health had been very good. She had one miscarriage and has three healthy children; her confinements were normal. She consulted me in September, 1933, for sore throat, post-nasal discharge and a husky voice. I was astonished to see the state she was in, though she made no complaint other than concerning her throat. Her face was puffy and intensely cyanotic, dark blue and blotchy. She was short of breath when sitting up in bed, and there was a generalized œdema of the whole body.

The patient was sent to hospital and swabs were taken from her nose and throat, but no Klebs-Löffler bacilli were found. After an X ray examination of the sinuses was made Dr. Hiller reported that she was suffering from subacute pharyngitis and infected tonsils, but that no sinusitis or no laryngeal lesion was present.

General examination revealed very little beyond generalized œdema, intense cyanosis of face and hands, and dyspnœa. The heart rate and rhythm were normal, and the blood pressure was normal. There was no œdema of the lung bases and neither the liver nor the spleen could be palpated. The patient's urine wsa scanty and concentrated, having a specific gravity of 1030 and showing a moderate cloud of albumin. The bowels were constipated. The blood count gave a hæmoglobin value of 90%, 6,000,000 red cells per cubic millimetre and 9,000 white cells, with normal differential count. The halometer reading was normal. There was no reaction to the Wassermann test and spectroscopic examination revealed no abnormal bands. An X ray picture of the chest revealed a somewhat bovine type of heart.

After a week or so in hospital the patient appeared to be going down hill. She had several alarming attacks, during which she sank into unconsciousness, her colour became almost black, her respiration was of the Cheyne-Stokes type, and her pulse rate was slow. She recovered from these attacks with disconcerting suddenness—at one minute being apparently near the point of death and the next minute sitting up in bed inquiring what the fuss was about. After the second week the red cells had increased to 8,000,000 per cubic millimetre. The patient was kept on a low caloric diet and given a course of "Salyrgan" and large doses of thyreoid extract. Adrenaline relieved the dyspnœa. The œdema was soon controlled and the patient's condition began gradually to improve on this treatment; at the end of two months her condition was comparatively normal.

Discussion.

A discussion of this case resolves itself into a consideration of the causes of cyanosis. The actual cause of cyanosis, of course, is an increase in the amount of reduced hæmoglobin, in any form, within the skin capillaries. This may arise centrally, when the heart is congenitally abnormal, when there are abnormal lung conditions and when methæmoglobinæmia or sulphhæmoglobinæmia is present, or peripherally, when the rate of blood flow through the skin diminishes sufficiently, owing to constriction of the arterioles. This may be due to a local drop in temperature or to central causes, and does

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on April 10, 1934.

not necessarily denote venous congestion. Dyspnœa is due to excess of carbon dioxide or to some abnormal acid in the respiratory centre. Thus cyanosis and dyspnœa are not always necessarily associated; for example, they are not associated in uræmia and congenital heart disease. The principal causes of cyanosis appear to be interference with diffusion in the lungs, pumping of unaerated blood into the arteries without passage through normal lung, and slowing of capillary flow (Langdon Brown).

Nature's method of compensating for defective oxygenation of existing blood corpuscles is by adding to their number. Thus with chronic cyanosis there is polycythæmia; and, conversely, polycythæmia causes cyanosis by slowing the capillary circulation. Taking these facts into consideration, the diagnosis in this case is against polycythæmia, and the subsequent history bears this out, since the patient's blood count is now normal. In one text book on the differential diagnosis of extreme cyanosis, no less than 62 causes were listed. In this case I do not propose to attempt to eliminate 61 of these causes, but merely to cut down the field for discussion.

Signs of congenital abnormality of the heart and local lung conditions were not present. No laryngeal signs denoted ulceration, obstruction or œdema; there was no history of drug addiction, and spectroscopic examination eliminated both methæmoglobinæmia and sulphhæmoglobinæmia. Diphtheria was eliminated, and also the angina of Ludwig. There was no evidence of pressure from an enlarged thyreoid gland. Subacute nephritis was suggested by the œdema, scanty urine and presence of albumin; but the only manner in which nephritis can cause cyanosis is in the last stages by œdema of the glottis or by cardiac failure. It appears that we are thrown back on the commonest of all causes of cyanosis, namely, failure of the right side of the heart, probably in this case due to myxœdema, perhaps to fatty infiltration, and relieved, first, by getting rid of the congestion and œdema and, secondly, by the loss of weight through restricted diet and thyreoid feeding. The patient's weight has been reduced from 129 kilograms (twenty and a half stone) to under 107 kilograms (seventeen stone), and she is still losing weight.

The patient's condition is at present as follows: Her resting pulse rate is 80, with regular rhythm. Her heart appears to be normal, though the reserve is limited somewhat. The heart size cannot be demonstrated on account of the thickness of the chest wall. The systolic blood pressures is 130 millimetres of mercury and the diastolic pressure 80. The hæmoglobin value is 80% (Sahli). The red cells number 5,050,000 per cubic millimetre. There is very slight œdema of the legs, and no albumin is present in the urine. The patient has septic teeth and tonsils and weighs 104 kilograms (sixteen stone eight pounds). There are no obvious signs of subthyreoidism: the skin is smooth, the hair and eyebrows are normal, and the pulse rate is not slow; the patient's mental outlook is normal and she has no feeling of cold. The metabolic processes do not appear to have become slower, and no skin rashes are present. (Read's formula for the estimation of the basal metabolic rate, I presume, relates only to cases of hyperthyreoidism. The rate works out in this case as +13·9.) There may have been a pituitary element in this case, but an X ray picture of the patient's skull revealed no alteration in the size of the *sella turcica*.

THE USE OF HEAT AND LIGHT IN VARIOUS CASES.

By E. PAYTEN DARK, M.B., Ch.M. (Sydney),
Katoomba, New South Wales.

Facial Paralysis.

A PROFESSIONAL man, who was suffering from a complete facial paralysis, consulted me on July 2, 1934. His general health was normal, except for a slightly raised blood pressure; he had been working hard for three years without any holiday, and a couple of days before the paralysis appeared he had been in a bitterly cold room for several hours.

From July 2, 1934, to July 17, 1934, he had complete rest, with the orthodox treatment; there was very slight improvement.

On July 17, 1934, he began a week of infra-red radiation, applied locally; each treatment was rather less than an hour, and he had one treatment every day. The improvement was dramatic; after three treatments movement of the facial muscles had improved fully 50%; at the end of the week the only trace of paralysis left was a very slight weakness of the muscles of the lip, noticed only when exaggerated contraction was attempted.

This case is of interest in view of the recent article by Dr. N. D. Royle in THE MEDICAL JOURNAL OF AUSTRALIA of September 22, 1934, on the "Conservative Treatment of Facial Paralysis", in which no mention is made of infra-red radiation.

W. Annandale Troup, in "Therapeutic Uses of Infra-Red Rays", which was very favourably reviewed in THE MEDICAL JOURNAL OF AUSTRALIA some months ago, states, on page 76, that he has not seen any case of Bell's paralysis fail to clear up rapidly under infra-red radiation.

Delayed Union of Fracture.

On January 23, 1934, a man was referred to me by a colleague. He had an ununited fracture of both bones of the leg at the junction of the middle and lower thirds. The fracture, which had been compound, had occurred five months before, and every effort had been made to induce union. The fracture was reduced under anæsthesia in the X ray room and put up with Smertz hooks on a Thomas frame with extension, and it had later been put up in plaster, all treatment being controlled by X rays, which showed excellent position but practically no callus formation, even after five months.

After consultation with his medical attendant he was given calcium with parathyreoid and "Radiostoleum", and he had a daily one-hour treatment with diathermy through the site of the fracture. On alternate days he had general ultra-violet radiation, the dose, of course, being increased according to his skin tolerance, the aim being to produce a mild erythema with each dose. This routine treatment was followed until the end of February, although after four weeks there appeared to me to be good union.

After treatment had ceased X ray examination revealed a good mass of firm callus.

Threatened Gangrene.

On December 7, 1933, I was called to see an old lady because of severe pains in the foot and the great toe. The toe was plum coloured and there were large patches of purple discoloration on the sole of the foot and on the heel; all the discolored areas were exquisitely tender. Two years before she had lost part of the other great toe as the result of a similar, but less extensive, condition.

She was sent into hospital, where the urine was found to contain 6% of sugar. The carbohydrates in her diet were greatly cut down, but that did not affect the sugar much. Later she was given one dose of five units of insulin, which was followed by restlessness, headache, and a feeling of great oppression. Two days later a dose of three units was tried, with similar results, so it was concluded that the sugar was due to a renal glycosuria.

When the old lady was admitted to the hospital several of my colleagues saw her, and the general opinion was that I would be amputating the foot in a few days. Probably the interference to the circulation was caused by an *endarteritis obliterans*.

As the great physiological effect of diathermy is to increase blood supply by a dilatation of small arteries and capillaries, I thought it was well worth while to try it. The electrodes were placed, one to the sole of the foot and the other to the back of the calf, and she had an hour's treatment each day. During the remainder of the time the foot was kept comfortably warm by hot water bags. During the first treatment the colour of the toe slowly changed from a sinister purple to a pleasing mauve.

For ten days she had treatments daily, and then for a week on alternate days. By the third treatment the toe and the other discolored patches became quite pink, while

she was having the diathermy, and were less and less cyanosed for the rest of the day; the pain also rapidly vanished. In just over two weeks the foot was quite normal and has remained normal to the present time.

Prophylactic.

A young man gave the history that three days before he had had intercourse with a girl; he took no precautions against venereal infection and afterwards found out that she was suffering from acute gonorrhœa. This was confirmed by bacteriological examination. He was naturally worried about the fate of his urethra. After that interval of time any ordinary form of prophylactic treatment did not appear to have any chance.

Cumberbatch and Robinson ("Treatment of Gonococcal Infections by Diathermy", 1925, page 10) state that a degree of heat which can be tolerated by the tissues of the body without harm is prejudicial or fatal to the gonococcus. Corbus and O'Connor ("Diathermy in the Treatment of Genito-Urinary Diseases, with Especial Reference to Cancer", 1925, page 30) state that a temperature of 108° F. will destroy gonococci in the tissues in thirty or forty minutes.

Normal tissue cells will stand up to 118° F. for longer periods without damage. Many types of gonococcal infection are quickly cleared up by diathermy, but so far no one seems to have had much success in acute urethritis in the male, the difficulty being to get the heat where it is needed. In this case there was as yet no actual sign of infection, and presumably the gonococci, if present, would only be in the anterior part of the urethra.

I passed a straight sound to the full length of the anterior urethra and connected it to one terminal of the diathermy machine and connected the other terminal to a large indifferent electrode placed on the patient's abdomen. The ampèrage was gradually increased until the urethra felt as hot as could be borne with comfort. He had a treatment of half an hour each day for five days and no infection developed.

Of course it is possible that there would never have been any infection in any case, and I record the history only as a suggestion that it might be worth while to investigate the possibilities of diathermy as a prophylactic when it is rather late to expect success from more ordinary methods.

Thrombosis.

At the beginning of November, 1933, a patient was referred to me from Sydney for treatment of thrombosis of the left internal saphenous vein.

In May of that year her uterus was curetted for a supposed miscarriage and she was in hospital for ten days. Her condition remained unrelieved, and in June a laparotomy was done by a gynæcologist and a four months ectopic gestation was found. Eleven days after the operation a thrombosis of the left internal saphenous vein occurred; ten days later there was a thrombosis of veins in the left arm. She was in hospital for eleven weeks, during which time the condition of the arm improved rapidly, but the leg was very little better—blue, stiff and swollen.

The surgeon thought that there was no chance of her walking in less than a year, but suggested to the husband, who was a medical practitioner, that he should consult me about the possibility of diathermy being of help. It seemed to me that diathermy, *plus* infra-red radiation, would be well worth trying, and I asked if she would come up to me for a few weeks, if possible.

At the time I began treatment the leg was quite useless, blue, cold, and lacking in epicritic sensibility to 12·5 centimetres (five inches) above the knee. The patient could get about with difficulty on crutches, so I arranged to give two treatments daily with a portable diathermy machine in her room, and one treatment of diathermy combined with infra-red radiation at my surgery. The diathermy was given by placing one electrode against the sole of the foot, and the second on the upper third of the thigh. The dose given was as much as the patient could tolerate with comfort, for about an hour.

During the treatment with infra-red radiation the whole leg was exposed to the rays; this caused a bright, widespread erythema, which lasted for a long time and, I believe, was of more benefit than the diathermy.

Improvement was excellent from the beginning, and after a fortnight sensation was normal and there was no cyanosis, except after the leg had been dependent for about two hours; muscular power was good, and the feeling of heaviness returned only with the cyanosis. At this stage I should have liked the patient to try walking a little with only a stick, but her consultant was not willing.

She had about three weeks' treatment from me and then returned to Sydney, where she continued with the diathermy once a day, but left off the infra-red treatment.

Six months after the beginning of the treatment she was walking normally and there was no hyperplasia of the tissues of the leg. At present, if she spends a long afternoon shopping in the city, the leg may get a little swollen, stiff and cold, but one treatment of diathermy gives relief.

I feel sure that if it had been practicable to keep up the three hours daily of diathermy and infra-red radiation, the time of treatment would at the least have been halved.

Conclusion.

This small group of cases may be of value in suggesting that in many varied conditions some form of electrotherapy may be of great service and should be considered by any doctor who wishes to do his best for his patients.

Reviews.

A SURVEY OF PSYCHOPATHOLOGY.

THE first edition of "Psychopathology: A Survey of Modern Approaches", by Dr. J. Ernest Nicole, was reviewed in the May 16, 1931, number of this journal; the present review concerns its second edition.[1] The principal additions deal with the works of Freud, with those of Jung, and with biochemical and physiological contributions, the eclectics and characterologists. New chapters have been written on ethnological and sociological evidence, the position in psychology, and applied psychopathology. The aim, as in the previous edition, has been to fill the gaps between text books of psychology and psychiatry by giving an adequate description of all schools. The author must be congratulated on his sincere and unbiased summary of divergent viewpoints, which include those of Morton Prince, Freud, Adler, Jung, Rivers, Watson, Kemp, Berman, Kretchmar, together with a host who receive honourable mention. The extent of the reading involved is shown by a very complete bibliography. Whilst it is easy to criticize the book on account of works which have been omitted, it is obvious that only a vastly larger edition could contain all.

The reader who leisurely turns over the pages may be intrigued along two lines of thought: first, that psychopathology is still in its youth, since there are so many conflicting alleged first principles; secondly, that the author should write a companion volume giving his own views as to the manner in which this maze of contradictions can be brought into a state of harmony. At present the reader is given the facts and is left to draw his own conclusions.

The book closes with four appendices which originally appeared in the *Journal of Mental Science*, but have now been revised. Appendix I deals with the concept of the ego in psychiatry and is largely based on the views of

[1] "Psychopathology: A Survey of Modern Approaches", by J. E. Nicole, L.M.S.S.A., D.P.M.R.C.P. and S., with foreword by W. H. B. Stoddart, M.D., B.S., F.R.C.P.; Second Edition; 1934. London: Baillière, Tindall and Cox. Demy 8vo., pp. 298. Price: 12s. 6d. net.

Freud. Whilst the presentation is good, the reader will probably agree with the concluding remark that "the whole subject is still somewhat obscure".

Appendix II is concerned with type psychology and its importance in mental hospital practice. The classification is based on Jung—thinking, feeling, intuitive, and sensational types of extroverts and intraverts. Dr. Nichols points out that a scientific study of personalities is essential, not merely to the welfare of patients, but also to the harmonious working of hospital staffs.

Appendix III deals with psychology and the herd instinct. After an able review of the literature, the conclusion contains a paragraph to which most students will admit acquiescence.

> We have so vacillated from side to side, picking up one theory and then dropping it for another, considering this and that view only to point at its insecurity, embarking upon a promising trail without the necessary time to follow it up, that we are hard put to enunciate any but vague and tentative conclusions.

It is certainly not the fault of the author that our knowledge is not crystal clear. There can be no hesitation in saying the book should be in the library of every one who has more than a casual interest in psychopathology and psychology. The reader will be grateful for a concise and well written summary by one who has obviously burned much midnight oil in its compilation.

INSTRUMENTAL INVESTIGATION IN HEART DISEASE.

"CLINICAL INVESTIGATION OF CARDIOVASCULAR FUNCTION", by Pachon and Fabre, is recommended to physicians, but we cannot agree with Pachon that it is a book for the general practitioner.[1] After reading it one can imagine Madame Curie's feelings as she contemplated the huge mass of pitchblende, but yet felt that there was in it somewhere something she had not possessed before. A wealth of variety of methods is described, but that very important aspect of cardio-vascular investigation, history-taking, is not mentioned. Indeed, the scope of the work would be shown better if to the title were added the words "by instrumental methods except the stethoscope". Moreover, electrocardiography and radiography are reviewed summarily, the sections devoted to these subjects occupying respectively twenty-five and eighteen pages. On the other hand, left lateral cardiography and oscillometry are discussed in fifty-two and one hundred and three pages respectively. The apparent discrepancy is explained by Pachon's belief that, in the investigation of the functional efficiency of the myocardium, the electrocardiogram is of little value, but that cardiography in the left lateral posture, oscillometry and oscillography are of the first importance. The heterodoxy of his views on electrocardiography makes the article on this subject of doubtful value to the practitioner, whilst the value of left lateral cardiography, although introduced in 1902 (by Pachon), has not become accepted by British physicians. Pachon's avowed ideal is instrumental measurement of biological phenomena. His invention of the oscillometer is a happy result. Nevertheless, the futility of trying to find a gauge for myocardial inefficiency in chronic heart disease by measuring the systolic contraction, wholly or in its phases, with the cardiograph, has been demonstrated already. It is nearly twenty years since one Australian investigator attacked this problem. He found himself stopped by the entanglements of the coronary arteries, the sufficiency of which is of such great importance for the integrity of the myocardium. The same obstacle likewise interferes with the use of the oscillometer as a gauge for the myocardium. However, the discussion in this book on the use of the latter instrument is useful, and physicians, although they must remain critical, can expect to find here subject for thought and investigation.

[1] "Clinical Investigation of Cardiovascular Function", by V. Pachon and R. Fabre, translated by J. F. Halls Dally, M.A., M.D., M.R.C.P.; 1934. London: Kegan Paul, Trench, Trubner and Company, Limited. Demy 8vo., pp. 263, with illustrations. Price: 15s. net.

CLINICAL ELECTROCARDIOGRAPHY.

WILLIAM EVANS has produced a very useful little book of simple type on the interpretation of electrocardiograms.[1] This is based on his association with the well known cardiac department of the London Hospital, and he has assembled an excellent collection of graphic records from the wealth of material available. The book is not a text book, nor is it an attempt to cover the field in an exhaustive fashion. It is designed to teach an orderly and logical method in unravelling these graphs, and is meant particularly for the undergraduate or post-graduate student who wishes to acquire the necessary knowledge and method. For this purpose the book may be warmly recommended; it is clear and simple, and, above all, brief, while containing all essentials. No student of its pages should fall into such elementary errors as diagnosing "cardiac failure" or "myocarditis" from an electrocardiogram; he should rather be helped along the right lines by a study of the beautifully reproduced records and their succinct descriptions.

PSYCHOLOGY.

IN recent years "of the making of books psychological there has been no end". Burridge's "A New Physiological Psychology", which strikes a new note, is one of the latest.[2] Now it is our lament that modern psychologists have vied with each other, not only in coining neologisms, but also in their use of an ever increasing complexity of argument. The result has been that the unfortunate reader, with his glossary at his side, wades painfully through many pages, knowing full well that very often he cannot grasp the author's exact meaning. Whilst he is prepared to admit that the writer is more intelligent than he is, he refuses to admit that such complexity of thought is either scientific or necessary. We have read and re-read time and again many of the paragraphs in this book without fully understanding their meaning.

The author is brilliant. He has an original message to deliver, a message of interest to psychologists, physiologists and psychiatrists. He has linked physiology and psychology in a way no other writer has done. He has spoilt his message by his over-use of neologisms and by has slavish adherence to the style of modern psychologists. What busy medical practitioner has the time to decipher a hidden message?

In brief, the theories advanced are these: From prolonged experiment with hearts, the author contends that central neurones and sensory end organs are "rhythmically acting colloidal systems" with two sources of potential energy, namely, "absorption reactions and changes in colloidal aggregation". The interactions between these two sources of energy are capable of infinite variation and "provide the dynamic energy for evoking responses". The explanation of many psychological phenomena (and especially Freud's teachings) in the light of these theories is of particular interest. The author's brilliancy and his fluent pen are worthy of a much better fate. He has written primarily for the research student, whose brain is agile enough to grasp quickly highly involved and highly technical arguments. As his theories are so interesting to psychiatrists, whose outlook tends to become more and more utilitarian, he should write in a much simpler style, and then will undoubtedly have many more readers.

[1] "A Student's Handbook of Clinical Electrocardiography", by W. Evans, M.D., M.R.C.P.; 1934. London: H. K. Lewis and Company, Limited. Medium 8vo., pp. 60, with illustrations. Price: 5s. net.

[2] "A New Physiological Psychology", by W. Burridge, D.M., M.A., with foreword by Sir Leonard Hill, M.B., LL.D., F.R.S.; 1933. London: Edward Arnold and Company. Crown 8vo., pp. 165, with illustrations. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 8, 1934.

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INHERITANCE OF RESISTANCE TO INFECTIVE DISEASE.

IF it were possible to find a community, either of men or of animals, that was free from disease usually caused by bacterial infection, the non-existence of disease would be due either to the absence of virulent microorganisms or to the powers of resistance to infection inherent in the individual members of the community. The community would be secure from disease only if the resistance of its individuals were permanent and if the individuals of succeeding generations were also resistant to infection. In the laboratory, in the practice of veterinary medicine, and in the practice of human medicine it is exceedingly difficult to assess the factors that bring about variations in immunity to bacterial infection. The effects of environment and of heredity are interwoven in an apparently hopeless entanglement. During recent years a great deal of investigation has centred round the inheritance of resistance or immunity to disease infection. A critical review of this subject has recently been published under the direction of the Statistical Committee of the Medical Research Council of Great Britain by Dr. A. Bradford Hill, of the London School of Hygiene and Tropical Medicine.[1]

First of all, Dr. Hill discusses briefly both congenital passive immunity and acquired active immunity, since they are both induced by non-genetic factors and since they both enter or may enter into much of the experimental work that he reviews. The experimental work is mentioned under two headings: "Genetic Resistance in Birds" and "Genetic Resistance in Mammals". In each section the work of different investigators is referred to in some detail and the significance of the findings in each instance is discussed. Apart from Dr. Hill's own conclusions, this summary of the literature will serve as a valuable source of reference. The first impression that Dr. Hill gives is that the subject is complex in the extreme. At the same time, in spite of many doubts and more uncertainties, some definite facts emerge. Dr. Hill assumes, and he is safe in assuming, that inheritance is to some extent a significant factor in the production of immunity. Clinical experience alone shows that one individual of a race or species differs from another in susceptibility to various diseases and, further, it may be taken for granted that it is possible in the laboratory to produce among animals strains that are resistant to a given microorganism. Although Dr. Hill makes the assumption that has been stated, he is careful to point out that "one must regard with doubt any experiment in which the acquirement of resistant host strains depends upon breeding from animals that have survived an infection. If the offspring of such matings are in fact found to be more resistant than a control sample of the same species, this finding, standing alone, would not be sufficient to justify the adoption of a genetic interpretation". In spite of the difficulty of excluding acquired active immunity in experimental work, it may be concluded that a true genetic immunity may sometimes be developed by selective breeding. In regard to the grade of

[1] "The Inheritance of Resistance to Bacterial Infection in Animal Species: A Review of the Published Experimental Data", by A. Bradford Hill. Medical Research Council of the Privy Council, Special Report Series, Number 196; 1934. London: His Majesty's Stationery Office, pp. 71. Price: 1s. 3d. net.

resistance that may be obtained, we cannot do better than quote the following passage from Dr. Hill's conclusions:

> As regards the degree of resistance that can be attained by the selection of favourable genetic factors within a host strain the evidence is somewhat conflicting. The term 'resistance', it will have been noted, has been used to cover a range of differences extending from a slight delay in time to death to the survival of a relatively high proportion of the infected individuals. It seems a little doubtful whether a group of animals all of which succumb to the experimental infection can be regarded as resistant in any effective sense, even though they live significantly longer than unselected controls. On the other hand, the differences between the selected and unselected groups in final mortality are in some cases (though by no means in all) very large. But so far there is no evidence that by selective breeding a strain can be obtained which is uniformly immune to a given bacterial infection, even when the test of immunity is only a single injection of a dose of a pathogenic bacterium that fails to kill 100 per cent. of controls.

The general conclusion is that, were the essential genetic factors better understood, it would be possible to develop a strain of animals solidly immune to a continuous risk of contact infection. Dr. Hill is justified in his view that on the present evidence there seems little hope of immediate success along these lins.

Much then remains to be done; and we naturally ask ourselves whether time and effort should be spent on attempting to make further progress. The story of the growth of knowledge in immunology shows that though many of the new doctrines and their attendant discoveries appeared at first to lead to simplification, they really did nothing of the sort. Fresh and complex avenues were opened up, and these in turn sometimes brought about other gains. No one can tell whither efforts to understand the genetic factors in immunology may lead. Apart from possible collateral discoveries made while this understanding is gained, ability to produce disease-resisting strains of animals would, it may be conjectured, lead to changes of considerable magnitude. The laboratory, veterinary medicine and human medicine have been mentioned. Ability to produce disease-resisting strains of animals would be of the greatest advantage in the laboratory, for much could be learned of the invasion of animals tissues by bacteria and of the response of tissues to the invasion. In veterinary medicine one has only to mention the possibility of breeding cows resistant to disease to visualize the enormous benefit that humanity would gain from such a procedure. When we come to human beings our imagination runs riot. At the risk of having to face a charge of mixing the fantastic with the scientific, we would refer readers to a picture of a world in which science unrestrained holds sway, drawn by Bertrand Russell in his book, "The Scientific Outlook". This is a picture of science as the master of man, instead of as his handmaiden. Science need never become the master of man. In any case there appears to be no reason why every effort should not be made to discover all the factors concerned in genetic immunity. Dr. Hill has earned the appreciation of all immunologists and other scientists; he has provided a most useful *point d'appui* for the future.

Current Comment.

THE HYPERTENSIVE HEART.

THE importance of the cardiac changes in arterial hypertension is well known, for, apart from the risk of cerebral accidents, the greatest danger to the hypertensive patient is centred round his heart. Clinicians appreciate the prognostic value of the cardiac signs, in particular the subjective disturbances of dyspnœa or pain, and the objective finding of hypertrophy. Here, too, pathologists agree, for autopsies on the subjects of cardiac failure show that the hearts that weigh over about 500 grammes may as a rule be safely set down as hypertensive unless some valvular lesion is present. The actual changes that occur in the heart muscle are, however, not so clearly understood; in fact, it is often difficult to say why the heart should have failed.

In a review of the careful histological investigation of twenty-seven hearts of the hypertensive type Victor Levine remarks that the term "chronic myocarditis" is a poor one in such cases.[1] This will be freely conceded; the term is quite outworn, for there is no true inflammatory basis for the changes. On the other hand, it is not easy to assume such an hypothesis as ischæmia with fibrosis in the face of the conspicuous hypertrophic reaction in the muscle. The literature gives no clear guidance, as Levine points out, and on this account this study was undertaken. All the hearts studied weighed about 500 grammes or more, and representative blocks were removed for microscopic examination; the tissues were stained with Van Gieson's stain and classified according to the degree of fibrosis found, particular attention being paid to the degree of sclerosis found in the arteries of various sizes.

[1] *Archives of Pathology*, September, 1934.

Levine cannot confirm the findings of some other workers who claim that the fibrosis is proportional to the amount of sclerosis in the coronary arteries. In only six hearts out of the total of twenty-seven was there found a serious degree of sclerosis in the large arteries; the arterioles were affected in a similar number. But in more than half the hearts examined any changes observed in the coronary arteries were not sufficient to account for the fibrosis found in the myocardium. It appears to be true, as pointed out by other writers, that the most severe degrees of myocardial fibrosis are found in those hearts that are the subject of serious and extensive arterial sclerosis, but in other groups the same proportional representation was not apparent. Levine quotes the work of Perkins and Miller, whose results paralleled his own, in that they found a thickened endocardium, many new elastic fibres, and numerous scattered scars due to shrunken and degenerate muscle fibres enclosed in and partly replaced by fibrous tissue. It is suggested that these changes are due to a defective blood supply, but there seems to be a curious discrepancy between the actual muscle changes and the demonstrable arterial ischæmia. It certainly is strange that microscopic fibrosis is apparently invariably found in the hypertensive heart, irrespective of the occurrence of definite vascular disease. No relation could be discovered between age, sex, weight of the heart, race, the blood pressure, the presence of syphilis, or the known cause of death, and the amount of fibrosis in the myocardium. What does this mean? It would appear that no constant anatomical basis can be discovered for this fibrosis. Where coronary sclerosis is demonstrated there is a proportionate degree of fibrous degeneration in the heart muscle, but there is a large group of cases in which this explanation is inadequate. Consequently it seems that some functional disturbance must be at work, presumably over a long period, and a clue to the explanation is furnished, in Levine's opinion, in the work of Ricker and others. According to this author, there is reason to believe that some nervous irritative stimulus to the terminal twigs of the coronary tree may cause constriction with stasis of the blood and eventually necrosis of the cells supplied. The initial stimulus might be supplied by spasm of a larger vessel, and the known clinical course of arterial hypertension gives support to such an explanation, for arterial spasms certainly occur in other parts of the body. This hypothesis is not accepted by all authorities, for while it is tempting to assume that a stasis causes the fibrous degeneration in the hypertensive heart in those cases in which it is not due to an actual ischæmia, it has been pointed out that possibly some additional unknown toxic factor may be present. Looking at the problem broadly, we must admit that we do not know the exact ætiology of arterial hypertension itself; it is not surprising therefore that some of the associated phenomena are yet obscure. This work serves to emphasize that heart failure occurs in hypertension, even though definite or obvious coronary sclerosis cannot be demonstrated. After all it is fortunate that in the practice of medicine we rely upon estimates of function rather than upon exact anatomical proofs.

THE USE OF ANTHELMINTICS.

RULE-OF-THUMB methods are employed by most medical practitioners in the treatment of helminth infestation. The ideal anthelmintic has not yet been discovered; until it is, more scientific and logical use must be made of the drugs that are available. The principles and theories of anthelmintic medication are outlined in a recent valuable paper by Maurice C. Hall.[1] Perhaps the most important statement in this paper is that the essential factor in anthelmintic medication is:

> Good judgment and experience in the physician, who, realizing the more or less poisonous nature of the necessary drugs, will administer them in just and reasonable proportions and under suitable conditions.

"Suitable conditions" are not only safe conditions, but conditions favourable to the eradication of the parasites.

Hall states that the medical practitioner should know: the habits and life histories of the parasites; the particular anthelmintic to be used against a particular parasite, and the proper dosage in individual cases; the effect on the patient; the most suitable purgatives, and when and how to administer them; the history and present condition of the patient; how to prevent reinfection. Whipworms are notoriously difficult to eradicate; this is due, not to the worms' resistance to anthelmintic drugs, but to their habit of living in the vermiform appendix and the most dependent part of the caecum, which are reached by very little of the drug. It is wise therefore to give small repeated doses of a relatively innocuous drug in the hope that some of it will come in contact with the worms in their sheltered retreat. A knowledge of the life history of the parasites is important. For example, the larvæ of certain worms, at one stage in their life history, wander through the host's tissues, where they are immune to any attack by an anthelmintic. In discussing anthelmintics, Hall points out that *in vitro* experiments have a limited field of usefulness. For example, alcohol kills worms *in vitro*; but, "if alcohol were an anthelmintic *in vivo* as well as *in vitro*, man should have been rid of his worm parasites ages ago". In regard to the absorption of anthelmintics, he points out that probably two-thirds of a therapeutic dose of thymol and, according to some workers, practically the whole of a dose of carbon tetrachloride are absorbed. He stresses the value of purgation in hurrying the drug through the intestine and allowing less time for its absorption.

These are a few only of the points discussed by Hall. The paper is wholly admirable and devoid of padding; it should be read by everyone who has to treat helminth infestation of any kind.

[1] *The Puerto Rico Journal of Public Health and Tropical Medicine*, June, 1934.

Abstracts from Current Medical Literature.

SURGERY.

Fenestræ and Pouches in the Broad Ligament and Strangulated Intra-abdominal Hernia.

ARTHUR B. HUNT (*Surgery, Gynecology and Obstetrics*, May, 1934) writes about the fenestræ and pouches in the broad ligament as an actual and potential cause of strangulated intraabdominal hernia. In recent years attention has been called to several cases of intestinal strangulation through defects in the broad ligament. A review of the subject was stimulated by encountering recently two cases in which an aperture of the broad ligament was found without strangulation. This form of internal hernia is rare and probably is the least common of the intraabdominal strangulated herniæ. Only thirteen authentic cases of strangulation through defects of the broad ligament were found in the literature, and only two cases were noted in which such defects were present but unassociated with strangulation. This condition, however, probably is more common than the reported cases would indicate. In cases in which pouches are the offending defects, congenital anomalies may be strongly suspected. Distension and distortion of the broad ligament from pregnancy or pelvic tumours seem the most likely factors in the production of fenestræ in that structure. Older *multiparæ* are almost exclusively affected, although nulliparous women are not immune. The Baldy-Webster operation may be looked upon as an ætiological factor. The anomalies occur without strangulation. Two such cases are reported. Although the condition has never been diagnosed before operation, it might conceivably be diagnosed. The treatment is comparatively simple. The defect should be enlarged or incised to liberate the bowel and then carefully closed by sutures. Removal of the adnexa may be preferable if the patient is an elderly woman or if the circulation is seriously impaired. The round ligament may be utilized to give a firmer repair of an aperture. The diagnosis of the obstruction is made and intervention takes place. The morbidity, however, in this series was high.

Diverticula of the Vermiform Appendix.

HAROLD C. EDWARDS (*The British Journal of Surgery*, July, 1934) states that between the years 1927 and 1931 almost 1,500 appendices were removed surgically at King's College Hospital. Of these, eight showed the presence of diverticula. No single case has been observed in the last 2,680 consecutive *post mortem* examinations at the hospital. Probably the frequent association of inflammation with diverticula accounts for the greater incidence in surgical records. In more than 5,000 examinations of the appendix by X rays no diverticula were found. This may be explained by the frequent occlusion of the lumen at the base of the appendix when diverticula are present at the distal extremity. The average age of patients at operation was thirty-four years and the average duration of symptoms seven years. In all cases reported the diverticula were multiple. Most cases are associated with chronic appendicitis. A fully formed diverticulum always lacks a muscular coat. Usually it represents a true hernia through a gap in the muscle coat. In a few instances the muscle has simply been thinned out over a distension of the lumen. These should be regarded as sacculations rather than diverticula. All of the author's specimens showed inflammatory changes. His findings all seem to show that these chronic inflammatory changes in the appendix precede the development of a diverticulum. The favourite site for diverticula is along the concavity of the appendix, where the vessels of the meso-appendix enter, although many are found on the opposite or convex margin. The mucosa is found frequently to have been thrown up into folds; probably this is due to the relative shortening of the muscular coat. The muscle coats themselves are greatly thickened. The author expounds a thesis to explain the pathogenesis. He postulates the primary tonic spasm of the longitudinal muscle fibres causing the mucous membrane to be thrown into folds. This also results in increasing the vascular gaps. Should spasm of the circular muscle occur, the potential cavity of the appendix is diminished and the tension within increased; hence the mucous membrane will seek an outlet and may become herniated through the vascular gap. There are no symptoms directly referable to the presence of diverticula and only very rarely may the condition be diagnosed radiographically. The treatment in all cases is appendicectomy.

Irradiation of the Parathyreoids in Generalized Osteitis Fibrosa Cystica.

MAX CUTLER AND SEWART E. OWEN (*Surgery, Gynecology and Obstetrics*, July, 1934) discuss the irradiation of the parathyreoids in generalized *osteitis fibrosa cystica*. The relationship between generalized cystic disease of the skeletal system and hyperfunction of the parathyreoid glands has received much attention in the recent medical literature. The experimental production of the disease in animals by the injection of parathormone, the recovery of patients suffering from multiple cystic bone lesions after parathyreoidectomy, and the frequent association of the characteristic bone lesions with hypercalcæmia and parathyreoid tumours leave little doubt as to the ætiological relationship between hyperfunction of the parathyreoid glands and generalized *osteitis fibrosa cystica*. A case of generalized *osteitis fibrosa cystica* associated with hypercalcæmia is reported in which clinical improvement took place following treatment by radiation of the parathyreoid glands. This observation and others recorded in the literature suggest the use of this method in cases in which surgical operation is contraindicated or in which parathyreoidectomy fails to effect a cure. Radiation of each parathyreoid area separately is suggested as a possible aid in determining the site of the adenoma before operation, a procedure which may render the exploration technically less difficult. Further observations along this line are needed. The relative merits of surgery and radiation as the essential treatment of parathyreoid adenoma associated with generalized *osteitis fibrosa cystica* must await further observations.

Hydatid Disease of the Brain.

HAROLD R. DEW (*Surgery, Gynecology and Obstetrics*, September, 1934) writes of hydatid disease of the brain. As in the case of all organs, infestation of the brain with echinococcal disease may be either primary or secondary. In the former the cysts are derived from hexacant embryos which, after passage through the liver and lungs, have been carried by the carotid arterial stream to the brain. Secondary cysts are metastatic and result from the sowing by the blood stream of scolices, derived from a fertile primary simple cyst which has ruptured into the left side of the heart. It is obvious, although the fact has not been universally recognized, that the pathological and clinical aspects of these two types are very different, and until this is appreciated much confusion must occur in their interpretation. Primary cysts occur almost exclusively in young children, are simple, and give rise to clinical syndromes similar to any other benign, non-infiltrating tumours, and are amenable to surgical operation. Secondary cysts are metastatic, occur almost exclusively in adults or adolescents, are secondary to an intracardiac rupture of a fertile primary cyst into the left side of the heart. They produce protean nervous manifestations, owing to their multiplicity, and invariably produce fatal results, not being amenable to surgery. The diagnosis of either type may be suspected in "hydatid" countries, but even there may depend on positive intradermal or serological reactions.

Suppurative Osteomyelitis of the Mandible.

LEO J. MILTNER AND J. J. WOLFE (*Surgery, Gynecology and Obstetrics*, August, 1934) describe the treatment of suppurative osteomyelitis of the mandible. The method of treatment of suppurative osteomyelitis of the

mandible described in this paper was given trial because of the unsatisfactory results which had been obtained by the use of the more conservative plans. A review and a discussion of the surgical treatment of osteomyelitis of the mandible is presented. A method of management for the chronic stage is described. The method embodies the following steps: Removal of the necrotic outer plate of the mandible as soon as possible during the early chronic stage, that is, twenty-one days after the onset. The necrotic bone is removed through a wide external approach and may be excised before it has separated spontaneously in the form of a sequestrum. In case of massive necrosis of both plates of the mandible sequestrectomy is delayed until involucrum has formed. In regard to removal of the teeth over the area of osteomyelitis, these teeth are always loose and bathed in pus, and in most instances their pulp tissue is necrotic. The next stage is exteriorization of the tooth sockets and partial resection of the alveolar process, with complete closure of the gum margins to prevent further drainage of purulent material into the oral cavity and immobilization of the jaw. The results of eight cases treated successfully by the new method of management are presented. The results have been very encouraging and are believed to be superior to those obtained by the more conservative treatment.

Extrapleural Pneumolysis with Paraffin Pack.

JEROME R. HEAD (*Surgery, Gynecology and Obstetrics*, August, 1934) tells of extrapleural pneumolysis with paraffin pack. This paper is a report of twenty-eight cases of pulmonary tuberculosis treated by extrapleural pneumolysis with paraffin pack. Extrapleural pneumolysis with paraffin pack has a definite place in the surgical treatment of pulmonary tuberculosis. The complications caused by the foreign body, which have deterred many from using the method, have been largely eliminated. When they do occur, they are rarely serious and rarely prejudice the patient's chances of recovery. This method has the great advantages of being simpler, safer, and less deforming than thoracoplasty and of making a strictly localized collapse of diseased lung without sacrificing vital capacity. For these reasons it can be used in a large group of cases in which all other methods are either impossible, are contraindicated or have failed. It has thus increased appreciably the number of patients amenable to collapse therapy. Whether or not it should ever be used in preference to thoracoplasty is still a subject of dispute. Sauerbruch and Brauer, the leaders of opinion in Germany, have recently changed their ideas and now believe it the most suitable operation for small apical cavities. It has been the author's experience that in cases in which the cavity does not extend lower than the fourth rib at the spine it is reasonably certain of producing the desired result. For larger cavities thoracoplasty is more certain. When the indications are doubtful, one may be influenced by the consideration that the lesser operation may suffice and that if it does not, a later thoracoplasty will be more certainly effective for the partial collapse already provided. Results from such secondary thoracoplasties have been so good that at present, when confronted with a very large apical cavity, the author uses a pack as a preliminary operation.

Intrathoracic Goitre.

FRANK H. LAHEY AND N. W. SWINTON (*Surgery, Gynecology and Obstetrics*, October, 1934) describe intrathoracic goitre. Any patient with mechanical interference to breathing should be suspected of having an intrathoracic goitre and should be subjected to an X ray examination of the mediastinum. In the presence of an intrathoracic goitre deviation or flattening of the trachea, either laterally or anteroposteriorly, together with the mediastinal shadow, can be demonstrated in practically all cases. Dilatation of the superficial thoracic veins should always make one suspicious of the presence of an intrathoracic goitre. Any goitre which is low-lying and which tends to become intrathoracic should be removed before it becomes intrathoracic. The introduction of a catheter between the vocal cords into the trachea makes the removal of large and difficult intrathoracic goitres infinitely more safe. Statistics are given showing the incidence of Grade I and Grade II intrathoracic goitres. Figures are also submitted showing the age incidence, sex incidence, the incidence of pre-operative and post-operative laryngeal paralysis, the incidence of tracheal deviation or pressure, the incidence of obstructive symptoms, the duration of the goitre, the incidence of hyperthyreoidism, the immediate post-operative complications and the mortality rate, together with the cause of death in 21 cases in a group of 1,086 patients operated upon with partly or completely intrathoracic goitres.

The Symptom Complex of Complete External Pancreatic Fistula.

ROBERT W. GARIS AND WALTER C. KERKEL (*Surgery, Gynecology and Obstetrics*, October, 1934) write about the symptom complex of complete external pancreatic fistula. The physiology of the external secretion of the pancreas is now rather well known, but the clinical syndrome and the pathological significance of the loss of this important secretion to the body are still generally unrecognized, if one can judge by the literature on the subject. It is suggested that complete exclusion of pancreatic juice from the intestine with loss by external fistula produces a highly characteristic clinical syndrome consisting of striking anorexia, nausea, intermittent vomiting, steatorrhœa, extreme exhaustion, anhydræmia and emaciation with lethal termination. A clinical example of this symptom complex is presented, corresponding in all important respects with the deleterious and fatal effects of complete pancreatic fistula in laboratory animals. It appears that partial or incomplete pancreatic fistula may in certain instances be associated with such symptoms as anorexia, weakness, loss of weight and steatorrhœa. Concomitant biliary principle and absence of trypsin or erosive properties in the fistulous fluid may readily lead to errors in diagnosis. The question is raised as to whether the exclusion of pancreatic juice from the intestine in some types of complete obstructive jaundice may not be the major factor in the rapidly fatal outcome, as, for example, in carcinoma of the ampulla of Vater and carcinoma of the pancreas. A short *résumé* of the more accepted methods of treatment in pancreatic fistula is presented.

One-Stage Lobectomy in Bronchiectasis.

A. TUDOR EDWARDS AND C. PRICE THOMAS (*The British Journal of Surgery*, October, 1934) discuss one-stage lobectomy for bronchiectasis and give an account of forty-eight cases in which this operation has been performed. They point out that lobectomy for bronchiectasis relatively confined to a single lobe of the lung has usually been carried out in two or more stages on account of fear of infection of the pleura and a mobile mediastinum, and that the mortality has been high. The authors describe the technique of the operation used by them. They point out that whenever extensive pleural adhesions do not prevent its accomplishment, they induce artificial pneumothorax one week before performing lobectomy. Four of the forty-eight patients died from conditions associated with the operation. Three died later, one from cerebral abscess five weeks after operation, two from tuberculosis of the other lung. In the two last-mentioned instances terminal complications occurred, but a large residual empyema was present on the operated side. One of these deaths occurred three months and the other nearly six months after operation. Of the remaining forty-one patients, six have some residual symptoms that are slight when compared with their original symptoms. Thirty-five patients are virtually symptomless. In the series lobectomy for malignant disease was not included, though in one instance a carcinoma, unsuspected before operation, was discovered. The authors conclude that lobectomy performed in one stage appears justifiable and should be the operation chosen for unilateral bronchiectasis.

Special Articles on Treatment.

(Contributed by request.)

LXIV.

POTT'S FRACTURE.

THE term Pott's fracture is somewhat loosely applied in practice to describe all those fractures in the region of the ankle that are produced by violent abduction of the foot. These fractures vary in type according to the intensity of the force applied and the length of time for which it acts. In the first type the external malleolus is twisted off, the line of fracture running from above and behind downwards and forwards; there may be no deformity at all, or the malleolus may be displaced somewhat outwards and slightly upwards, and there may be faint lateral displacement of the astragalus. In the second type the pull of the deltoid ligament tears off the internal malleolus, which breaks transversely at its junction with the shaft of the tibia; and this permits not only further lateral movement of the astragalus, but also tilting of its upper articular surface, which is no longer horizontal, and its upper outer border tends to insinuate itself into the angle between the tibia and fibula. The lateral displacement of the external malleolus is more pronounced and the internal malleolus also moves somewhat laterally. This is the type that produces the typical abduction eversion deformity of the foot with the lower end of the shaft of the tibia prominent beneath the stretched skin, or the skin may even give way at this point, rendering the fracture compound. In the third type, in addition to the above-mentioned deformity, a triangular fragment is broken off the posterior part of the lower articular surface of the tibia and this allows the whole foot to drop backwards, the triangular fragment itself also moving backwards and usually somewhat upwards.

Reduction of the fracture should be carried out at the earliest possible moment. The practice prevalent in some quarters, of waiting for the excessive swelling to subside, has nothing to recommend it and only renders the ultimate reduction more difficult and the liability to any redisplacement greater. The application of plaster of Paris, preferably to the unpadded skin, is the only effective method of retaining the fragment in the corrected position. A box splint is, if anything, slightly worse than useless; not only will it fail to maintain reduction, but it stands a very good chance of producing a pressure sore on the back of the heel.

Since the displacements vary only in degree, we can lay down a standard method of reduction and fixation for all cases. The displaced fragments must be made to retrace their steps; the heel must be hooked forwards and will pull with it the triangular fragment which has been broken off the posterior part of the articular surface of the tibia. The lateral malleolus must be pushed inwards and it in its turn will force the astragalus and medial malleolus back to their normal position.

In some books one sees the advice given to put the foot up in the inverted position. This is neither necessary nor desirable. The movement of inversion is carried out mainly at the subastragaloid joint, and the lateral displacement of the astragalus and of the external malleolus can still be present in spite of well marked inversion of the foot; and such a position must not be substituted for the actual pushing back of the external malleolus into position. Even in the presence of numerous blebs, the plaster can still be applied to the unpadded skin; it is only necessary to prick the blebs with a sterile pair of scissors and allow the fluid to escape and then paint the skin with methylated spirits before applying the plaster. The fragments must be held in the corrected position whilst the plaster is being applied, and this involves the only difficulty which has to be overcome. If an assistant attempts to hold the part in the corrected position whilst the surgeon applies the plaster, the subsequent procedures resolve themselves into a struggle between the surgeon and his assistant. If the former gains the day, his assistant's hands are frequently pushed off the part and redisplacement may easily occur; if the assistant refuses to allow himself to be pushed out of the way, an accurately fitting plaster cannot possibly be applied. We must therefore have some mechanical means of supporting the part satisfactorily with no excess of hands in the way.

Whichever method be adopted, the deformity is first reduced by hooking the heel forward with the fingers of one hand whilst with the other hand the laterally displaced lateral malleolus is pushed medially, the astragalus being forced over at the same time. Preparations are then made to apply the plaster. One method which requires no special apparatus is to employ loops of bandage about 5·0 centimetres (two inches) wide to hold the position whilst the plaster is applied. The patient is placed with his foot projecting over the end of the table, one loop passes round the surgeon's neck and beneath the patient's heel, the other loop passes across the front of the patient's leg, just above the ankle joint, the other end hangs down towards the floor and in it the surgeon places his foot, as in a stirrup. The combined effect of the two pulls is to pull the heel forward and to push the leg backwards in relation to the foot. The maintenance of the corrected lateral displacement depends on the surgeon's own hands. With these slings in position the unpadded plaster of Paris casing is employed. It is always advisable to use a large slab extending down the posterior aspect of the leg and thence along the sole of the foot, reaching to the extremity of the toes. After this slab is applied the plaster is completed by the use of several bandages applied in the ordinary circular fashion. It is important that the plaster should extend to the tips of the toes on the plantar surface. On the dorsum the phalanges only should be left exposed to view, in order to watch subsequently the state of the circulation. A common error is to carry the plaster only as far distally as the mid-tarsal region. Such a plaster gives rise to considerable discomfort, as it allows marked œdema of the forepart of the foot to occur. Whilst the plaster is setting, the surgeon moulds it vigorously with his hands so that it forms a close fit about the bony prominences of the ankle joint. When setting is complete, the bandage loops are cut through at the point where they emerge from the plaster and one additional plaster bandage is employed to cover in the points where they have emerged through the plaster and to give a neater appearance to the whole structure.

In the use of this method care must be taken lest the sling which passes across the front of the leg may make too strong pressure in this situation, for in such event there is definite danger of the formation of a pressure sore.

Preferable to the method described above is some form of skeletal traction, preferably with a Schmertz hook. Whatever may be the differences of opinion as to the long-continued use of skeletal traction, no objection can be raised to its temporary employment for a matter of thirty minutes. This temporary traction is best carried out by means of a Schmertz hook used in conjunction with a Watson-Jones apparatus, but since such an apparatus is not generally available to all practitioners, I will content myself by describing a perfectly efficient method of carrying out the principle associated with the use of this apparatus without describing the actual use of the apparatus itself.

The patient, when anæsthetized, must be placed with the leg hanging over the end of the table and with the knee bent to a right angle. A ring bolt is temporarily screwed into the floor vertically beneath the patient's limb. After manual reduction of the deformity the Schmertz hook is inserted into the heel and the traction cord attached to the hook can then be passed through the ring bolt and made fast, steady continuous traction thus being given. The counter-traction is furnished by the posterior surface of the lower thigh lying on the table. Small wisps of cotton wool soaked in friar's balsam are wrapped round the points of the hook, where they penetrate the skin, and the plaster is applied in the usual fashion with the employment of a posterior slab. This is the ideal method, and nothing then interferes with the quiet unhurried application of a close-fitting, well-moulded casing. As soon

as the plaster is set, the hook is withdrawn. Of course, in moulding the plaster care must be taken that the heel is carried well forward, that there persists no lateral displacement of the lower end of the fibula, that the foot is at right angles to the leg and neither inverted nor everted. The plaster should also be well moulded into the sole in order to preserve the natural shape of the arch of the foot. Just as the plaster is setting, the plaster knife should be taken and the whole casing split down the middle line in front from top to bottom. When this is done, the edges of the plaster will gape till they are separated by about one-sixteenth of an inch, and if there be much subsequent swelling this gap will spontaneously widen still more during the next day or two.

Circulation of the toes must be closely watched. No alarm need be felt if they become somewhat dusky in colour; the danger signals which indicate that the plaster is too tight are blanching of the toes, particularly of the little toe, or loss of sensation in any toe. In such cases the plaster which has already been split can be prized apart at the line of the split, and this gives as much additional space as is desired. Many patients have a corn on the dorsum of their little toe previous to their accident and sometimes complain of pressure by the plaster in this situation. This should not be met by trimming up the plaster so as to expose the little toe, but by prizing the edges of the distal part of the plaster slightly apart.

On the following day an X ray examination should be made through the plaster to determine the position of the fragments and the correctness of the reduction. An X ray examination through plaster is usually perfectly satisfactory for determining the position of fragments, but is quite valueless in the determination of the presence of union. After the plaster has been applied a few days' rest in bed should be enjoined in order to allow the reaction to settle down and for any excessive swelling to subside.

Subsequent treatment can follow one of two methods. Either the plaster is left untouched for four weeks and then bivalved into medial and lateral halves, or else within seven days a walking iron is added so that all the weight is transmitted to the leg well above the site of fracture and the patient is allowed to walk about at will in this iron. In my experience the latter is the preferable method, since it keeps muscle and bone atrophy and joint stiffness at a minimum. In approximately ten weeks the plaster and walking iron are removed and an X ray examination is made to determine the presence of union. If this is satisfactory, weight-bearing is then permitted, but, except in the case of children, the inner border of the sole and heel of the boot should be raised six millimetres (a quarter of an inch) and an outside iron and an inside "T" strap fitted to the boot; this appliance should be employed for a further three or four months.

If this precaution be not taken, gradual redisplacement of the fragments is liable to occur and the limb, which at the moment of its removal from the plaster is in excellent position, will gradually reassume the original deformity. If a walking iron has not been employed and the plaster has been bivalved, at the end of four weeks the limb should be removed every day from its splint and active movements and exercises should be carried out, assisted by massage, to diminish any œdema which may be present. In this event also protected weight-bearing can usually be permitted at the end of eight weeks.

Solid bony union is the usual result in the fracture of the fibula and in the posterior lipping fracture of the tibia. Quite frequently bony union fails to occur in the fracture of the medial malleolus and only a close fibrous union takes place. This, however, is not productive of any disability and does not appear to interfere with the stability of the joint in the least and never requires operative interference. In general the prognosis of Pott's fracture is excellent, provided good reduction be secured. In some few cases, however, in spite of perfect reposition of the fragments, traumatic arthritic changes gradually occur in the ankle joint, leading to a chronically painful ankle. This is more apt to happen when there has been the posterior lipping fracture of the tibial articular surface. Such a happening cannot be predicted and nothing can be done to prevent its occurrence. Indeed these arthritic changes may not come on till some years have elapsed from the time of injury.

The chief point in the treatment of these fractures, on which emphasis must be laid, is the fact that plaster of Paris is the only efficient splinting material and that the box splint has no better place in the treatment of these fractures than has the long Liston in fractures of the femur. Disasters have occurred in the past from the use of plaster of Paris, but there need be no fear if the plaster be split from top to bottom along the anterior aspect at the time of application and a careful watch be kept on the circulation.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Sydney Hospital on October 18, 1934. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Polyposis of the Colon.

DR. HAROLD RITCHIE showed a girl, aged twenty years, who was suffering from polyposis of the rectum and colon. The patient had had two attacks of what was diagnosed as ulcerative colitis. Dr. Ritchie pointed out that cases of apparent ulcerative colitis in young people without fever should always be regarded as possible examples of rectal polyposis; the patients should always be examined with the sigmoidoscope. He also drew attention to the fact that this condition in a large number of cases took on malignant changes. There were two main types of polyposis: (i) a familial variety, and (ii) the other arising as the end result of a true ulcerative colitis.

Multiple Mesenteric Cysts.

Dr. Ritchie also showed a child of four years with multiple mesenteric cysts, some chylous and some containing clear fluid, probably congenital in origin. The pre-operative diagnosis had been tuberculous peritonitis.

Myeloid Leuchæmia.

DR. A. W. HOLMES À COURT showed a boy, aged three years, who was admitted to hospital on October 5, 1934. The history was of progressive enlargement of the abdomen during the preceding four months, associated with emaciation and gradual depreciation of health.

On examination there was obvious pallor. The spleen was grossly enlarged, causing great abdominal distension. The crenated anterior border of the spleen could be felt in the right iliac fossa. The liver was not palpable, nor were the lymphatic glands enlarged. A blood count revealed the following information:

Red cells, per cubic millimetre	4,570,000
Hæmoglobin value	74%
Colour index	0·9
White cells, per cubic millimetre	153,700
Neutrophile cells—	
(*a*) Mature	22·50%
(*b*) Myelocytes and metamyelocytes	34·75%
Myeloblasts	6·25%
Eosinophile cells—	
(*a*) Mature	4·75%
(*b*) Immature	5·50%
Basophile cells—	
(*a*) Mature	7·00%
(*b*) Myelocytes	14·50%
Lymphocytes	4·75%

Poikilocytosis, anisocytosis, nucleated red cells (five normoblasts per hundred white cells) were present. Diffuse polychromasia and punctate basophilia were noted. The fragility of the red cells was within normal limits. The Kahn and Casoni tests gave no response. The bilirubin content of the plasma was less than 0·5 unit.

The examination of the blood established the diagnosis as myeloid leuchæmia. The splenic enlargement was of unusual degree.

Cranio-Pharyngioma.

Dr. Holmes à Court also showed a girl, aged fifteen years, who had complained for one month of headache and loss of vision affecting particularly the right eye. She was subnormal mentally and showed evidence of pituitary dysfunction, stunted growth, "pudding face" and girdle obesity. Menstruation had not appeared, although breasts and sexual organs appeared normally developed.

The head was globoid in shape. Optic disks showed papilloedema proceeding to secondary optic atrophy on the right side. Owing to defective vision, perimetry was unsatisfactory. No other localizing signs were demonstrable on examination of nervous symptoms.

Radiographic examination of the cranium revealed erosion of the *sella turcica* with a ring of calcification above it. The cranial sutures were widened and the appearance of the cranial bones was characteristic of increased intracranial tension.

Schilder's Disease (Encephalitis Periaxialis Diffusa).

Lantern slides and photographs of the brain were exhibited from a male patient, aged seven years, who had suffered from Schilder's disease (*encephalitis periaxialis diffusa*). The full pathological and clinical record in this case will, it is hoped, be published *in extenso* at a later date.

Polycythæmia.

DR. G. C. WILLCOCKS showed a man, aged thirty-four years, who was suffering from polycythæmia. The patient gave a history of having been quite well until October 16, 1933, when, after a meal, he had an attack of dizziness, nausea and vomiting. He was then admitted to hospital. He was found to be suffering from arterial hypertension, cardiac hypertrophy and aortic dilatation. His blood serum did not react to the Wassermann test.

On October 27, 1933, his erythrocytes numbered 8,320,000 per cubic millimetre, the hæmoglobin value was 151% and the colour index 0·9.

On November 10, 1933, the erythrocytes numbered 9,060,000 per cubic millimetre, the hæmoglobin value was 156% and the colour index was 0·86.

On September 15, 1934, the erythrocytes numbered 9,580,000 per cubic millimetre, the hæmoglobin value was 176% and the colour index was 0·9.

Dr. Willcocks said that phenylhydrazine hydrochloride had been given in varying doses without definite effect. The patient was in hospital in order that more intensive treatment might be undertaken.

Cystic Disease of the Tibia.

Dr. Willcocks also showed a woman, aged forty-nine years, who complained of pains and weakness in the legs. The pain was that commonly associated with the sciatic nerve. X ray examination revealed the presence of a cyst situated anteriorly in the middle third of the tibia, probably due to *osteitis fibrosa cystica*. Changes discovered in the skull, the lower dorsal and lumbar vertebræ suggested early *osteitis deformans*. The patient gave no reaction to the Wassermann test. The blood calcium content was 11·6 milligrammes *per centum*, and the blood cholesterol 200 milligrammes. Examination of the blood revealed a colour index of 0·057 and the changes in the cells were those characteristic of a secondary anæmia.

Dr. Willcocks explained that the patient was being treated by the administration of "Radiostoleum" and calcium lactate. He said that the chief interest in this case was that changes both of *osteitis fibrosa cystica* and of *osteitis deformans* were present.

Auricular Fibrillation.

Dr. Willcocks's third patient was a man, aged thirty-eight years, who complained that his heart beat rapidly, that he got short of breath and that he had a choking feeling. On examination the patient was found to have a slow pulse rate, 60 to the minute. The pulse was irregular and the irregularity was apparently irregular. At this stage it was difficult to decide between a sinus arrhythmia and fibrillation of the auricles with a slow rate. The heart was found to be enlarged and a mitral murmur was detected. Electrocardiographic examination revealed: (i) auricular fibrillation, (ii) a ventricular extrasystole, (iii) left branch bundle block. A history of rheumatic fever twenty years previously was elicited. Dr. Willcocks pointed out that the slow pulse rate made diagnosis extremely difficult until an electrocardiogram was taken.

Neurosyphilis.

Dr. Willcocks also showed a patient who was suffering from neurosyphilis. The patient had complained of weak legs for a period of two years. Examination revealed an uncertain gait and small pupils that did not react to light. The knee jerks were brisk and ankle clonus was present on the right side, and the plantar reflex was occasionally extensor in type. Both blood serum and cerebro-spinal fluid reacted to the Wassermann test. The patient had spastic paraplegia and some ataxia without any pronounced mental changes. Dr. Willcocks said that in view of its supposed greater penetration into the nervous system the anionic bismuth preparation, iodobismuthate of quinine, was being used in the treatment of this patient.

Osteitis Deformans.

Dr. Willcocks's last patient was a woman, aged sixty-one years, who complained of loss of sight in the right eye, of a large head and of bowing of the femora. The patient's systolic blood pressure was 230 and the diastolic pressure 110 millimetres of mercury. The blood serum did not react to the Wassermann test. X ray examination revealed the skull changes of Paget's disease, and also thickening and bowing of the upper extremity of the femur. Ophthalmological examination revealed an arteriosclerotic fundus and an exudate in the right macular region. Dr. Willcocks commented on the combination of Paget's disease with pronounced arteriosclerosis and high blood pressure.

Coronary Sclerosis and Myocarditis.

DR. WILFRED EVANS showed a woman, aged sixty-nine years, who had complained for three months of giddiness, faintness and breathlessness and for two years of pain in the left side of the chest and left arm after exertion.

On examination the systolic blood pressure was 180 and the diastolic pressure 100 millimetres of mercury. The apex beat was in the fifth intercostal space, 11·25 centimetres (four and a half inches) to the left of the middle line. The cardiac sounds were soft and of poor tone, and the rhythm was tic-tac in character. There was no œdema or liver enlargement. The electrocardiograph exhibited a perfectly normal tracing in Lead I. In Lead II the *T* waves were flattened and in Lead III arose slightly below the line and were deeply inverted. Dr. Evans said that this tracing was one upon which it would be difficult with certainty to give a diagnosis of organic disease, although the *T* waves in Lead III were rather suggestive. The Wassermann test gave no reaction. The case was an example of the type of coronary sclerosis met with commonly in late middle life. The physical signs were often not very definite, but in this case there was definite pendulum rhythm with a characteristic history.

Syphilitic Aortitis and Myocarditis.

Dr. Evans showed a man who had complained of breathlessness on exertion since July, 1930. Physical examination showed that the apex beat was in the fifth space, 12·5 centimetres (five inches) from the mid-line. The heart sounds were of poor tone, the rhythm was of the pendulum type, and the second aortic sound was accentuated. The systolic blood pressure was 140 and the diastolic pressure was 95 millimetres of mercury. The chest was very emphysematous. X ray examination revealed marked cardiac enlargement with diffuse aortic dilatation. The Wassermann test gave a complete positive reaction.

Electrocardiographic examination of the heart showed that the rate was 110 per minute. The rhythm was regular. The *P* wave was upright in Leads I and II and inverted in Lead III. The *P-R* interval was 0·16 second. The *Q R S* complex was upright in Leads I and II and diphasic in Lead III. The *T* wave was upright in Leads I and II and deeply inverted in Lead III.

Dr. Evans said that the only feature suggestive of disease was the very deep inversion of *T* in Lead III. Otherwise nothing abnormal was noted. The patient had been given a full course of "Salvarsan" and mercury after preliminary treatment with iodides by mouth and after mercurial inunction. The diagnosis was syphilitic aortitis without clinical signs of involvement of the aortic valve itself. This case illustrated the axiom that in an enlarged heart, without evidence of high blood pressure or aortic valvular disease, syphilis should be suspected.

Right Branch Bundle Block.

Dr. Evans then showed a man, aged sixty years, who complained of shortness of breath and pain in both shoulders.

On examination the patient's chest was seen to be emphysematous. The systolic blood pressure was 160 and the diastolic pressure 90 millimetres of mercury. The apex beat was in the fifth space, 10·0 centimetres (four inches) from the middle line. The heart sounds were of poor tone and the rhythm was tic-tac. The reaction of the urine was acid; its specific gravity was 1025. It contained sugar, but no albumin. The glucose tolerance test yielded the following figures: 108, 126, 212, 218, 183. The Wassermann test gave no reaction. X ray examination of the chest revealed slight cardiac enlargement, mainly to the left side. Electrocardiographic examination revealed right branch bundle block.

Complete Heart Block and Branch Bundle Block.

Dr. Evans also showed a man, aged sixty-five years, who complained of breathlessness and precordial pain on exertion. The patient's systolic blood pressure was 260 and his diastolic pressure was 120 millimetres of mercury. The radial vessel wall was very thickened. The apex beat was in the fifth intercostal space, 12·5 centimetres (five inches) from the middle line. The heart sounds were of poor tone, a systolic murmur was heard all over the heart, and a third sound could be detected at all areas. The cardiac rate was 44 per minute. On X ray examination the heart was found to be enlarged in all diameters, and the enlargement affected chiefly the left ventricle, with some dilatation of the right side. The lungs were the site of venous congestion. The electrocardiograph revealed complete heart block and right branch bundle block. The Wassermann test gave no reaction.

Complete Heart Block.

Dr. Evans's next patient was a man, aged sixty-two years. The patient complained of breathlessness on exertion. Physical examination revealed the classical signs of complete heart block. There was no definite cardiac enlargement, but a loud systolic murmur was heard at the apex. The systolic blood pressure was 200 and the diastolic pressure was 90 millimetres of mercury. The Wassermann test yielded no reaction. X ray examination showed that the heart was not enlarged. The bronchial glands were enlarged. Pleural adhesions were present at both bases.

Electrocardiographic examination revealed complete heart block. The ventricular rate was 30 per minute. The auricular rate was 110 per minute. The rhythm was regular. The *P* waves were upright throughout. They were completely dissociated from the ventricular rhythm. The *Q R S* complexes were upright in Leads I and II and inverted in Lead III. They were very notched and widened to 0·12 second. The *T* waves were inverted throughout.

Dr. Evans said that it was noteworthy that in 1931, for a period of some months, the signs of heart block clinically and, as shown by the electrocardiograph, completely disappeared.

Mixed Flutter and Fibrillation.

Dr. Evans also showed a man, aged fifty-nine years, who had complained of extreme breathlessness on exertion since December, 1933. When he was first seen in April, 1934, physical examination revealed an apex beat in the fifth intercostal space, 10 centimetres (four inches) from the middle line; the heart rate was 160 per minute and the rhythm was regular. Electrocardiographic tracings showed typical auricular flutter. At a later date the heart rhythm became irregular and tracings then showed auricular fibrillation. The systolic blood pressure was 130 and the diastolic pressure was 90 millimetres of mercury. The Wassermann test yielded no reaction. X ray examination revealed diffuse dilatation of the aorta, but the heart shadow was of normal size. Treatment consisted in the administration of *pulvis digitalis foliæ*, 0·12 gramme (two grains) three times a day. Dr. Evans said that this maintained his heart rate at 70 to 80 per minute. The patient had no previous history of rheumatic fever, there was no evidence of hyperthyreoidism, and the cause of the fibrillation was probably myocarditis.

Secondary Dextrocardia.

The last patient shown by Dr. Evans was a man, aged sixty-three years. He had complained of cough and copious yellow expectoration for eight weeks. He was being treated for syphilis at the Board of Health. On examination there was dulness, cavernous breathing and pectoriloquy over the right side of the chest and the cardiac apex was felt well to the right. X ray examination revealed a gross displacement of the mediastinum and heart to the right side, due to atelectasis of the right lung. The appearance suggested a right bronchial stenosis, possibly carcinomatous rather than due to fibrosis. There was no evidence of tuberculosis in the emphysematous left lung. Electrocardiographic examination revealed poorly developed *T* waves, but otherwise no abnormality was present.

Rodent Ulcer.

DR. GEORGE BELL showed a male patient, aged sixty-one years, a tramway ganger, who gave a history on March 20, 1933, that for eighteen years he had had a rodent ulcer on the side of his nose. The ulcer had disappeared for two years after treatment with radium. On its reappearance it was again treated with radium and it disappeared for a short time. It then increased slowly in size and radium was used again, but without success. In July, 1930, the lesion was excised, the right *ala nasi* being removed for a distance of 3·1 centimetres (one and a quarter inches) up towards the root of the nose. The patient was admitted to hospital on March 20, 1933, for a plastic operation on the nose.

On March 29, 1933, the first stage of the operation was performed. A tube graft was prepared from the right side of the neck. The graft was about 15·0 centimetres (six inches) long and stretched from the angle of the mandible to below the clavicle.

On April 26, 1933, the second stage of the operation was performed. The distal end of the tube graft was swung round and sutured in position on the right side of the nose, the proximal end being left attached to the neck. Small pieces of cartilage from the second left costal cartilage were inserted into the right side of the nose.

On June 14, 1934, the third stage of the operation was performed. The portion of the graft between the neck

and the right cheek was removed, the graft being left attached to the nose and cheek.

At the time of demonstration the result was excellent.

Compound Fractures Treated by Wiring.

DR. ARCHIE ASPINALL demonstrated two patients who had been treated for severe compound fractures. The first was a young man who was injured in a motor accident and had both bones of the leg broken, complete loss of continuity of the tibia being caused and there being more than a dozen separate fragments. These were carefully readjusted and held in position by four separate phosphobronze wires encircling the bones.

The wires had been removed, healing by first intention had been secured, and the patient was able to walk well, with no shortening and good bony alignment.

This case will be reported more fully later.

The second patient had a compound comminuted supracondylar fracture of the humerus with dislocation of the elbow. It had been treated with open operation and encircling wire. Healing occurred by first intention, and with the exception of some limitation of flexion, a perfect functional result had been obtained, full pronation and supination being present.

Dr. Aspinall explained in detail the method of treatment and said that the sooner such fractures were operated on after the injury, the better the results.

Plastic Operations on the Face.

Dr. Aspinall showed two cases of successful rhinoplasty. One of the patients had suffered from lupus and had been treated with X rays and radium and had developed malignant disease of the nose requiring surgical treatment. The previous extensive scarring of the skin of the face increased the difficulty of the operation. A tube graft had been used.

The second patient was a young man who had total destruction of the nose from congenital syphilis. A new nose had been formed by means of flaps from the forehead providing skin on the inner and outer side of the nose with cartilage between.

Photographs were shown indicating the various stages of the operation.

The third patient had malignant disease involving the whole lower lip. The original condition was a small epithelioma of the lip four years previously, which had been treated by radium but did not react favourably. The patient was referred to Dr. Aspinall for surgical removal of the growth. A tube graft had been prepared, to be used in the making of a new lower lip after the removal of the growth and dissection of the glands of the neck.

The cases illustrated plastic operations coming within the scope of the general surgeon.

Hydatid of the Lung.

DR. HOWARD BULLOCK showed a woman, aged twenty years, who had been admitted to hospital on September 19, 1934. She was engaged in domestic duties and gave a history of pain in the chest over the upper part of the sternum and in both sides of the chest; the pain was more severe on the left side. The pain became more acute and occurred at irregular intervals. The pain was present first three months ago. The patient said that she had had a cough for the last nine months. The cough had been most severe one month previously and yellowish white sputum was present in small quantities. The patient vomited about two pints of brown offensive fluid about one month before admission to hospital; no "grape skins" were present in the vomitus. The patient was breathless on exertion and this symptom had been more pronounced during the previous month.

Physical examination revealed no deformity of the chest. Expansion was even. Decreased vocal fremitus was present at the base of the left lung. On percussion a dull note was heard at the left base posteriorly. Diminished breath sounds and an occasional friction rub were heard at the left base. The heart was not displaced. The liver was thought to extend to the sixth intercostal space at the right mammary line.

Dr. Bullock said that certain special tests had been carried out. X ray examination revealed a large hydatid cyst at the left base and a smaller cyst in the middle field of the right lung. A full blood count was made and an eosinophile count of 0·5% was recorded. The Casoni, Wassermann and Kahn tests all yielded no reaction. The complement deviation test for hydatid disease gave a complete positive reaction. A positive reaction was also obtained with the precipitin test for hydatid disease.

On September 21, 1934, left thoracotomy was performed, portions of five ribs being removed. The operation of partial lobectomy was performed. On October 6, 1934, the wound was completely healed. X ray examination on October 10, 1934, showed that the lung was still collapsed. Section of the cyst wall showed the characteristic lamination of a hydatid ectocyst.

In discussing the case Dr. Bullock pointed out that the cyst was moving freely with the lung and had formed no pleural attachments. He said that it would have been difficult to have eradicated the cyst thoroughly without performing lobectomy.

Loose Body in the Knee Joint.

Dr. Bullock's second patient was a young man, a clerk, aged twenty-five years, who had been admitted to hospital on March 14, 1934. The patient gave a history that six years previously his knee became locked while he was playing football and became swollen. A diagnosis of synovitis was made. On several occasions the joint became locked. On each occasion a lump appeared on the medial side and the patient used to replace the lump himself.

On examination it was found that the movements of the joint were quite normal; a slight degree of synovitis was present. X ray examination revealed the presence of a loose body in the joint.

On March 16, 1934, the medial meniscus was removed from the joint. The meniscus was found to be partly calcified and separated from its posterior attachments. On March 27, 1934, the patient was discharged from hospital cured. The specimen was a tough, partly calcified meniscus.

Dr. Bullock drew attention to the fact that the anterior and lateral attachments of the cartilage were quite normal and that the posterior portion of the cartilage was lying free. He also laid emphasis on the rarity of calcification in a meniscus.

Carcinoma of the Rectum and Uterine Fibroids.

Dr. Bullock also showed a woman, aged forty-seven years, who was admitted to hospital on November 15, 1934, and on whom abdomino-perineal excision of the rectum and hysterectomy were performed at one sitting for carcinoma of the rectum and uterine fibroids.

The patient gave a history of diarrhœa for eleven months. At first she passed blood and mucus and had about ten motions a day. For the two months prior to her admission to hospital she passed five motions a day; during this two months no blood or mucus was present. For eleven months the patient had suffered from hæmorrhoids. Her appetite was good and she had not vomited. She had no urinary symptoms, nor any symptoms referable to pelvic organs.

On examination by abdominal palpation no abnormality was discovered. On rectal examination a mass was palpable in the anterior wall of the rectum, about five centimetres (two inches) from the anus. The mass was not fixed; it was about 7·5 centimetres (three inches) in circumference. On vaginal examination no abnormality could be discovered.

On November 17, 1933, hysterectomy and abdomino-perineal resection of the rectum were performed. At operation the uterus was found enlarged, with two pedunculated fibroids.

On November 19, 1933, the colostomy was opened.

On January 2, 1934, the patient got out of bed, and on January 23 she was discharged from hospital. Pathological examination revealed carcinoma of the rectum with no involvement of the glands.

In discussing the case, Dr. Bullock said that in spite of the magnitude of the operations the patient had suffered comparatively little shock.

Thoracoplasty for Pulmonary Abscess.

Dr. Bullock then showed a woman, aged twenty years, who was engaged in domestic duties and who was admitted to hospital on March 4, 1932.

The patient gave a history that eight months before her admission to hospital she suffered from pneumonia and pleurisy on the right side, following confinement. Since then she had suffered from severe cough with copious foul-smelling sputum (a quarter of a pint *per diem*). She also had severe hæmoptysis and had lost weight.

On examination the patient was seen to be very pale. On the right side of the chest diminished movement was noted, the percussion note was dull and bronchial breath sounds and râles were heard. X ray examination revealed consolidation of the right lung with a central translucency suggesting an abscess of the lung. Fluid was present at the right base. A blood count revealed a secondary anæmia. The sputum was examined for tubercle bacilli, but none were found.

On March 11, 1932, evulsion of the right phrenic nerve was performed. Following this operation the patient's condition improved to a certain extent, but she was readmitted to hospital on May 19, 1932, in much the same condition as before, but also suffering from melancholia.

On June 15, 1932, the lung was drained and partial thoracoplasty was performed. At operation portions of the lower seven ribs were resected subperiosteally. On July 6, 1932, thoracoplasty was performed, portions of the remaining ribs being removed.

In discussing the case, Dr. Bullock said that the patient had been an invalid and was unable to get about owing to weakness. She had constant cough and expectoration of pus. Since the operation she had gained over two stone in weight. She now had no shortness of breath and no cough, and was singing in a choir.

Osteomyelitis of a Lumbar Vertebra.

Dr. Bullock's next patient was a boy, aged fifteen years, who had been admitted to hospital on December 7, 1933. He gave a history of having suffered for three days from pain in the right lumbar region; he had several severe attacks of pain. The pain would sometimes shoot into the groin, and the urine contained albumin.

Physical examination revealed tenderness in the right renal angle. The abdomen was slightly distended. There were no palpable masses. A diagnosis of osteomyelitis of the lumbar vertebra was made.

Dr. Bullock gave details of the special examination and tests that had been made, as follows:

1. Leucocyte counts were carried out on the following dates with the following results:

December 9, 1933	10,600
December 9, 1933	16,880
December 10, 1933	17,320
December 12, 1933	21,880
December 20, 1933	22,680
December 28, 1933	22,920
January 11, 1934	17,760

2. Repeated microscopic urinary tests revealed the presence of only an occasional pus cell.
3. The Casoni test gave no reaction.
4. The von Pirquet test gave no reaction.
5. X ray examinations were made on several occasions. On December 11, 1933, no bony deformity was discovered in the spine and the patient was kept at rest between sandbags. On December 20, 1933, the chest was found to be clear and no elevation of the diaphragm was found. On January 17, 1934, almost complete collapse of the second lumbar vertebra was noted. Commencing involvement of the third lumbar vertebra suggested the presence of osteomyelitis. On June 20, 1934, the condition was still active. On August 19, 1934, the lesion showed no advance.

In regard to treatment, Dr. Bullock said that the patient had been in a plaster jacket since January 18, 1934. A number of applications had been made. On August 20, 1934, a bone graft, taken from the shaft of the tibia, had been inserted and a jacket immediately applied. The jacket was taken down and reapplied on October 8, 1934; the wounds healed by first intention. In conclusion, Dr. Bullock pointed out that a feature of the case was the use of hyperextension to prevent dorsal deformity.

Stricture of the Urethra after Traumatic Rupture with Resulting Recto-Urethral Fistula.

Dr. Bullock's last patient was a youth, aged sixteen years, whom he had first seen in May, 1934.

The patient gave a history that he had fallen on to a rail fence four years ago. He was said to have extravasation of urine and later was sent to Sydney. In four years nineteen operations under general anæsthesia were performed. The patient complained of general ill-health and that he passed fæces *per urethram* at each defæcation.

On examination in May, 1934, the patient looked extremely ill. A suprapubic tube was draining away filthy urine. There was an impermeable stricture of the urethra in the region of its membranous portion, and at this point the catheter passed into the rectum. Intravenous pyelography by "Uroselectan" revealed considerable dilatation of the right ureter and pelvis of the kidney.

On May 23, 1934, left inguinal colostomy and external urethrotomy were performed, the stricture was excised and the ends were brought together after the method described in THE MEDICAL JOURNAL OF AUSTRALIA of November 23, 1918. The opening in the rectum was closed. A catheter was tied in for a week and it was replaced by another for a week. On trying to pass a third catheter, it slipped back into the rectum—the fistula had reformed.

On June 30, 1934, the *sphincter ani* was dilated, a quadrilateral area about 3·75 by 3·75 centimetres (one and a half by one and a half inches) was dissected from the anterior rectal wall. Its attachments to the part from which the stricture had been removed were carefully preserved. Its upper end was stitched to the proximal part of the urethra and its lower end to the distal part of the urethra, and then sewn over a tied-in catheter in the form of a tube. The hole in the rectum was repaired. Gauze was packed high up between the newly formed urethra and the rectum and left for seven days. The catheter was removed in ten days.

On July 21, 1934, approximately three weeks later, a 13/16 sound was passed with ease and then the colostomy and suprapubic wounds were closed.

On August 14, 1934, a 13/16 sound was passed with ease, and also on September 28, 1934.

Dr. Bullock pointed out that the patient had gained about three stone in weight and appeared to be in excellent health. He was apparently not feeling the damaged right kidney and had no stricture, the transplanted rectal mucosa apparently having joined up readily with the urethral lining.

(To be continued.)

ANNUAL MEETING, MELBOURNE, 1935.

IN connexion with the annual meeting of the British Medical Association to be held in Melbourne in 1935, it is proposed to hold an exhibition of the hobbies of medical men and women.

Such hobbies as drawing, painting, photography, craftwork of various kinds, and the collection of stamps, coins, butterflies, native weapons *et cetera* would be suitable for exhibition; many others will no doubt suggest themselves.

It is hoped that by the hearty cooperation of the medical profession an interesting and representative exhibition of special interest to overseas visitors may be displayed.

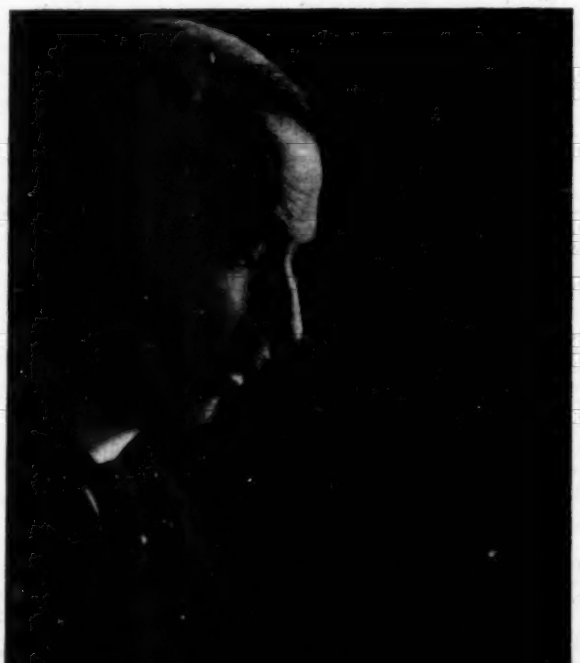
Any medical man or woman from any State in the Commonwealth who proposes to be present at the meeting and who would be prepared to take part in the Hobbies Exhibition is invited to communicate without delay with Dr. H. C. Colville, Medical Society Hall, Albert Street, East Melbourne, C.2.

Obituary.

CHISHOLM ROSS.

THE late Dr. Chisholm Ross, whose death was recently recorded in these pages, will be remembered by the older graduates of the University of Sydney as a teacher who, grudging neither time nor effort, gave of his best to the seeker after knowledge and as one to whom appeal in the difficulties of practice was never made in vain.

Born on October 29, 1857, Chisholm Ross was the son of the late Colin Ross, one of the early settlers in the Inverell district, New South Wales. Here Chisholm Ross was born. To think of the Ross family is to think of the Earls of Ross and the chieftainship of Clan Anrias or Ross. The history of this clan is of interest, particularly to Scotsmen. Rival claims were made for the chieftainship of the clan, and many held that Andrew Ross, the grandfather of Chisholm Ross, was entitled to wear the triple eagle's feather in his bonnet—the symbol of a highland chief. Chisholm Ross was the fourth child and the second son in a family of ten. His education was started at the Inverell school and later he was sent to The King's School, Parramatta. On leaving school, he went back to his father's estate to help him in his many pastoral and agricultural activities. He was trained as a wool expert and took his share in the hard life of the bush worker. Though this was not to be his life's work, the interest engendered by it never left him. In later years, when he was superintending the establishment of the mental hospital at Kenmore, he published some papers on agriculture, devoting particular attention to the making of ensilage. Botany claimed his attention, and he studied both grasses and forestry; he was concerned also with the economic aspects of these subjects.



Once it had become evident that medicine was to claim him, Ross went to Edinburgh to study. He graduated in 1883 as Bachelor of Medicine and Master of Surgery; in 1886 he became a Doctor of Medicine. He was also one of the first to be admitted to the *ad eundem* degree of Doctor of Medicine in the University of Sydney. On his return to Australia Ross entered the mental hospital service of New South Wales as assistant medical officer at Gladesville to the late Eric Sinclair. The present status of psychological medicine in New South Wales is probably due more to Eric Sinclair than to any one man, and without doubt Sinclair had an influence on Ross. At the same time Frederick Norton Manning, who preceded Sinclair as Director-General of the Insane and who was both an effective administrator and a wise psychiatrist, was Ross's friend and counsellor. His friendship and his wisdom were an inspiration to Ross. Ross had been in the department but a year or two when he was given charge of the mental hospital at Newcastle. At this time the superintendency of this hospital carried with it the post of port health officer. From Newcastle he was transferred to Goulburn to superintend the establishment of a new hospital at Kenmore. He remained at Kenmore until about 1900, when he was sent to Callan Park. After staying at Callan Park for a few years he left the department and took up private practice. It was at this period in his career that Ross became lecturer in psychological medicine at the University of Sydney. He was also appointed visiting medical officer to the Reception House, Darlinghurst. He was a quiet lecturer—he had none of the tricks of the orator, but relied on clarity and on sincerity. He could discuss the minutiæ of a subject without being diffuse and could in comparatively few words cover a wide range without omitting essentials. He sought to interest his students in the subject. His statement at the end of the course, that those who had listened to him might come to him for advice should difficulties arise, was not an empty one. He was always ready to help in a perplexing situation. This was done in an unobtrusive fashion. Some might count it a fault that he was no elaborate window-dresser. Had he sought prominence or pushed himself, he might have become what is known as a popular figure; but he would not have retained the admiration and respect of his fellows as he did. The qualities that he showed to his students were manifested throughout his whole professional life. Those who were privileged to know him in his private life found that he had, as one of them has written, "a subtle something in character, disposition and temperament which endeared him to all". "His consideration for the feelings of others was so delicate that rough handling of his sentiments could only emanate from a coarse and indelicate mind . . . . If ever man lived up to the highest standard of nobility of sentiment, it was he. He walked uprightly and worked righteousness and spoke the truth in his heart"!

LIONEL JOSEPH ROBERTSON.

WE regret to announce the death of Dr. Lionel Joseph Robertson, which occurred on November 3, 1934, at Subiaco, Western Australia.

WILLIAM THEODORE HODGE.

WE regret to announce the death of Dr. William Theodore Hodge, which occurred on November 20, 1934, at Derby, Western Australia.

Public Health.

POLIOMYELITIS IN NEW SOUTH WALES.

THE Infantile Paralysis Committee (New South Wales) desires to inform medical practitioners that the incidence of cases of infantile paralysis occurring in the Sydney metropolitan area is such as to constitute an epidemic. Forty-five (45) cases have been seen by the medical officer of the Infantile Paralysis Committee during the past two months, and convalescent serum has been administered to nineteen (19) patients. There have been three deaths.

Practitioners are requested to notify all cases to the Department of Health, and are informed that if they require assistance, such as a consultation or a bedside examination of the cerebro-spinal fluid and treatment by means of convalescent serum, they should communicate with the Medical Officer and Secretary, Infantile Paralysis Committee, "Locarno", 141, Macquarie Street, Sydney (telephones: BW 6409 by day, FL 2641 by night).

The following short *résumé* of the early symptoms of infantile paralysis is presented in order to refresh the memories of practitioners who have not seen a case since the last epidemic.

1. In the typical history there is a prodromal stage some two days to one week before the onset of any tenderness of the muscles or loss of power of the muscles. These symptoms may be an intense headache, feverishness, drowsiness, nausea and vomiting. There may be only the symptoms of the "common cold": running nose, sore throat and feeling of being "out of sorts".

2. Early "spinal" or preparalytic symptoms a day or so before the onset of paralysis are as follows: headache, drowsiness if left alone or irritability if disturbed, pain in the back and in the legs, and soreness and pain on attempting to bend the head.

3. Early signs are: presence of the spine sign, elicited on trying to approximate the head to the knees, rigidity of the neck, Kernig's sign, and tenderness of the muscles and hyperæsthesia.

Research.

THE WILLIAM GIBSON RESEARCH SCHOLARSHIP FOR MEDICAL WOMEN.

MISS MAUD MARGARET GIBSON has placed in the hands of the Royal Society of Medicine a sum of money sufficient to provide a scholarship of the yearly value of £292, in memory of her father, the late Mr. William Gibson, of Melbourne. The scholarship is awarded from time to time by the Society to qualified medical women who are subjects of the British Empire, and is tenable for a period of two years, but may in special circumstances be extended to a third year. The next award will be made in June, 1935.

In choosing a scholar the Society will be guided in its choice "either by research work already done by her or by research work which she contemplates. The scholar shall be free to travel at her own will for the purpose of the research she has undertaken".

There is no competitive examination, nor need a thesis or other work for publication or otherwise be submitted. The Society has power at any time to terminate the grant if it has reason to be dissatisfied with the work or conduct of the scholar.

Applications should be accompanied by a statement of professional training, degrees or diplomas, and of appointments, together with a schedule of the proposed research. Applications must be accompanied by testimonials, one as to academical or professional status and one as to general character. Envelopes containing applications *et cetera* should be marked on top left-hand corner "William Gibson Research Scholarship", and should be addressed to Mr. G. R. Edwards, Secretary, Royal Society of Medicine, 1, Wimpole Street, London, W.1, and be received not later than Saturday, June 1, 1935.

Correspondence.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

SIR: The Council of the Medical Benevolent Association of New South Wales has issued an appeal to the members of the medical profession in New South Wales for additional financial support to enable it to give each beneficiary a present at Christmas time. The help given apart from this suffices merely for the bare necessaries of life, and at this season of the year it is felt that something more is called for.

The Council therefore appeals to all members of the medical profession in New South Wales for contributions to a fund for providing Christmas comforts and cheer to the unfortunate members of the profession and their dependants on its list.

All contributions should be sent to Dr. E. S. Littlejohn, Honorary Treasurer, The Medical Benevolent Association of New South Wales, 135, Macquarie Street, Sydney.

Yours, etc.,

ALFRED W. CAMPBELL,
Chairman.

November 21, 1934.

CORRIGENDUM.

SIR: In an address to the British Medical Association (New South Wales Branch) I stated that one of our colleagues had received a gunshot fracture of the thigh and that he had been put on a Thomas splint by Vance.

I have been informed that the credit of the correct treatment belongs to Dr. T. W. van Epen, who was assisted by Dr. Harold T. Marsh, of Kempsey.

I regret the error and request you to publish this correction.

Yours, etc.,

J. COLVIN STOREY.

185, Macquarie Street,
Sydney,
December 1, 1934.

FOCAL INFECTIONS.

SIR: In your issue of November 10 Dr. Montgomerie Paton suggests that I may have been trying to steal his thunder. We discussed the matter together after I had read Handley's book. Personally I am not able to criticize Dr. Paton's views, as there seems so much speculation as to the secretions which Schafer has named chalones, hormones and autocoids. Of what their reaction is on one another I do not think any of us have the remotest idea. Dr. Paton's view is that cancer is due to some loss of balance in these secretions. My contention, drawn largely from the work of Handley, is that the blockage of the lymphatics, which Handley has shown to cause proliferation of the papilli, is the basic factor from which overgrowth starts. It is quite obvious that if lymphatic blockage causes hypertrophy, it was lymphatic drainage that prevented it. Neither Paton nor Handley has arrived at that very simple deduction. To neither has it occurred that the epithelium lining the walls of the lymph tubules must offer some sort of selective action in collecting the lymph exuding from the capillary walls. I shall look forward with pleasure to reading Dr. Paton's latest views on the subject. Although up to the present I have not been able to follow his line of reasoning, views expressed by a man who is sincere are always worthy of consideration.

Yours, etc.,

SYDNEY PERN.

12, Collins Street,
Melbourne,
Undated.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1933*, of Queensland, as duly qualified medical practitioners:

Corin, Kathleen Ruth, M.B., Ch.M., 1924 (Univ. Sydney), Rockhampton.
Grey, William Scoboria, M.B., 1929 (Univ. Sydney), Brisbane.
Lawrance, Kenneth George, M.B., 1929 (Univ. Sydney), Southport.
Rigano, Domenico, M.D., 1924 (Turin), Ingham.

Books Received.

HUMAN STERILITY: CAUSATION, DIAGNOSIS, AND TREATMENT: A PRACTICAL MANUAL OF CLINICAL PROCEDURE, by S. R. Meaker, M.D.; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 287, with illustrations. Price: 18s. net.

THE DIABETIC LIFE: ITS CONTROL BY DIET AND INSULIN: A CONCISE PRACTICAL MANUAL FOR PRACTITIONERS AND PATIENTS, by R. D. Lawrence, M.A., M.D., F.R.C.P.; Eighth Edition; 1934. London: J. and A. Churchill. Demy 8vo., pp. 232. Price: 8s. 6d. net.

NEW LIVES FOR OLD: HOW TO CURE THE INCURABLE, by J. E. Barker, with an introduction by Sir Herbert Barker; 1934. London: John Murray. Crown 8vo., pp. 384. Price: 7s. 6d. net.

Diary for the Month.

DEC. 11.—Tasmanian Branch, B.M.A.: Branch.
DEC. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 13.—New South Wales Branch, B.M.A.: Branch.
DEC. 13.—Victorian Branch, B.M.A.: Council.
DEC. 14.—Queensland Branch, B.M.A.: Branch Annual Meeting.
DEC. 18.—Tasmanian Branch, B.M.A.: Council.
DEC. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 21.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. C. Fortune (B.M.A.) has been appointed Resident Pathologist at the Perth Hospital, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiii, xiv, xv.

BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, NEW SOUTH WALES: Resident Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
METROPOLITAN INFECTIOUS DISEASES HOSPITAL, NORTHFIELD, SOUTH AUSTRALIA: Resident Medical Superintendent.
MOOROOPNA HOSPITAL, MOOROOPNA, VICTORIA: Senior Resident Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Resident Medical Officers, Resident Pathologist.
THE ROYAL SOCIETY OF MEDICINE, LONDON, ENGLAND: William Gibson Research Scholarship.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons, Post-Graduates.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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